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410
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     $0.71  Estimated cost this search
     $0.71  Estimated total session cost   0.198 DialUnits

File 410:Dialog Comm.-of-Interest Newsletters 2008 /Mar
       (c) 2008 Dialog

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? begion 5,73,155,399
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     $0.22  TELNET
     $0.22  Estimated cost this search
     $0.93  Estimated total session cost   0.313 DialUnits

SYSTEM:OS  - DIALOG OneSearch
  File   5:Biosis Previews(R) 1926-2008/Aug W5
         (c) 2008 The Thomson Corporation
  File  73:EMBASE 1974-2008/Sep 04
         (c) 2008 Elsevier B.V.
  File 155:MEDLINE(R) 1950-2008/Sep 05
         (c) format only 2008 Dialog
  File 399:CA SEARCH(R) 1967-2008/UD=14911
         (c) 2008 American Chemical Society
*File 399: Use is subject to the terms of your user/customer agreement.
IPCR/8 classification codes now searchable as IC=. See HELP NEWSIPCR.

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? e aufrentsch marco ?

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E1        2  AUFREISSPHANOMENE
E2        1  AUFREITER
E3        0 *AUFRENTSCH MARCO ?
E4        5  AUFRERE
E5       13  AUFRETEN
E6        2  AUFRETENDE
E7        1  AUFRETENDEM
E8        2  AUFRETENS
E9       16  AUFRICHT
E10       1  AUFRICHT G
E11       1  AUFRICHTBAR
E12       3  AUFRICHTEFEDER

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E9        1  AU=FRENTSOS J.A.
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E11       2  AU=FRENTZ B
E12       1  AU=FRENTZ B.

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               4  AU=FRENTSCH, MARCO
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E10       1  AU=ROTHE N VINGE
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E4        1  AU-THIOSULFATE
E5        1  AU-TRANS-Y-CAROTENE
E6        2  AU-006
E7        1  AU-006 1-(2-METHYLPHENYL)-4- (3-HYDROXYPROPYL)
E8        3  AU-1
E9        1  AU-1 CELL LINE (HOMINIDAE)
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E11       1  AU-1 LIKE FORMS
E12       1  AU-116

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E11      10  AU=THIEL B A
E12       1  AU=THIEL B G

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? s (s1 or s2 or s3) and (cd40 or cd40L ro cd154 or cd40(w)ligand)
              14  S1
               4  S2
             107  S3
           35372  CD40
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          550185  LIGAND
           16639  CD40(W)LIGAND
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? rd s4
      S5       3  RD S4  (unique items)
? t s5/3/all

 5/3/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2008 The Thomson Corporation. All rts. reserv.

0019901393   BIOSIS NO.: 200700561134
Identification and isolation of murine antigen-reactive T cells according
  to CD154 expression
AUTHOR: Kirchhoff Dennis; Frentsch Marco; Leclerk Patrick; Bumann
  Dirk; Rausch Sebastian; Hartmann Susanne; Thiel Andreas; Scheffold
  Alexander (Reprint)
AUTHOR ADDRESS: Deutsches Rheuma Forschungszentrum Berlin, Immunomodulat
  Grp, Charitepl 1, D-10117 Berlin, Germany**Germany
AUTHOR E-MAIL ADDRESS: scheffold@drfz.de
JOURNAL: European Journal of Immunology  37 (9): p2370-2377 SEP 2007 2007
ITEM IDENTIFIER: doi:10.1002/eji.200737322
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English


 5/3/2     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2008 Dialog. All rts. reserv.

16630900   PMID: 16186818
  Direct  access to CD4+ T cells specific for defined antigens according to
CD154 expression.
  Frentsch  Marco;  Arbach Olga; Kirchhoff Dennis; Moewes Beate; Worm
Margitta; Rothe Martin; Scheffold Alexander; Thiel Andreas
  Clinical  Immunology  Group,  Deutsches Rheuma-Forschungszentrum, Berlin,
Germany.
  Nature  medicine (United  States)  Oct  2005,  11  (10)  p1118-24,  ISSN
1078-8956--Print   Journal Code: 9502015
  Publishing Model Print-Electronic
  Document type: Journal Article; Research Support, Non-U.S. Gov't
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  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: MEDLINE; Completed


 5/3/3     (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2008 American Chemical Society. All rts. reserv.

  140286138    CA: 140(18)286138g    PATENT
  Method for detecting and isolating antigen specific T lymphocytes
  INVENTOR(AUTHOR): Frentsch, Marco; Rothe, Martin; Thiel, Andreas
  LOCATION: Germany,
  ASSIGNEE: Deutsches Rheuma-Forschungs Zentrum Berlin
  PATENT: PCT International ; WO 200427428 A1  DATE: 20040401
  APPLICATION: WO 2003EP9354 (20030822) *EP 200290300 (20020823)
  PAGES: 42 pp.  CODEN: PIXXD2  LANGUAGE: German
  PATENT CLASSIFICATIONS:
    CLASS:  G01N-033/569A
  DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ;
CA; CH; CN; CO; CR; CU; CZ; DK; DM; DZ; EC; EE; ES; FI; GB; GD; GE; GH; GM;
HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV;
MA; MD; MG; MK; MN; MW; MX; MZ; NI; NO; NZ; OM; PG; PH; PL; PT; RO; RU; SC;
SD; SE; SG; SK; SL; SY; TJ; TM; TN; TR; TT; TZ; UA; UG; US; UZ; VC; VN; YU;
ZA; ZM; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ  DESIGNATED REGIONAL: GH; GM; KE
; LS; MW; MZ; SD; SL; SZ; TZ; UG; ZM; ZW; AT; BE; BG; CH; CY; CZ; DE; DK;
EE; ES; FI; FR; GB; GR; HU; IE; IT; LU; MC; NL; PT; RO; SE; SI; SK; TR; BF;
BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD; TG
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           35372  CD40
            3387  ANTI(W)CD40
           35372  CD40
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         5955266  DETERMIN?
         4374224  EXPRESS?
          384777  CD4?
          210228  CD8?
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           35372  CD40
            3387  ANTI(W)CD40
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        3779758  DETECT?
         384777  CD4?
         210228  CD8?
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 10/3/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2008 The Thomson Corporation. All rts. reserv.

16963642   BIOSIS NO.: 200200557153
Characterization of soluble CD40 ligand released from human activated
  platelets
AUTHOR: Jin Yinzhu; Nonoyama Shigeaki (Reprint); Morio Tomohiro; Imai
  Kohsuke; Ochs Hans D; Mizutani Shuki
AUTHOR ADDRESS: Division of Human Ontogeny and Childhood Development,
  Graduate School, Tokyo Medical and Dental University, 1-5-45, Yushima,
  Bunkyo-ku, Tokyo, 113-8519, Japan**Japan
JOURNAL: Journal of Medical and Dental Sciences  48 (1): p23-27 Mar., 2001
2001
MEDIUM: print
ISSN: 1342-8810
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English


 10/3/2     (Item 2 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2008 The Thomson Corporation. All rts. reserv.

16616423   BIOSIS NO.: 200200209934
Direct effects of CD40L on CD40+AML blasts: Proliferation, self renewal,
  rescue from apoptosis and production of cytokines
AUTHOR: Aldinucci Donatella (Reprint); Poletto Dalisa (Reprint); Nanni
  Paola (Reprint); Degan Massimo (Reprint); Rupolo Maurizio (Reprint);
  Pinto Antonio (Reprint); Gattei Valter (Reprint)
AUTHOR ADDRESS: Nucleo di Ricerca Clinica e Laboratoristica in Ematologia,
  Centro di Riferimento Oncologico, IRCCS, Aviano, PN, Italy**Italy
JOURNAL: Blood  98 (11 Part 1): p589a November 16, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of
Hematology, Part 1  Orlando, Florida, USA  December 07-11, 2001; 20011207
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract;  Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English


 10/3/3     (Item 3 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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16558296 BIOSIS NO.: 200200151807
CD40 ligand accumulation during storage of human platelet concentrates: Implications for transfusion complications
AUTHOR: Vanderlinde Elizabeth (Reprint); Kaufman Julia; Phipps Richard; Blumberg Neil (Reprint)
AUTHOR ADDRESS: Transfusion Medicine, Univ. of Rochester Medical Center, Rochester, NY, USA**USA
JOURNAL: Blood 98 (11 Part 2): p112b November 16, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 2 Orlando, Florida, USA December 07-11, 2001; 20011207
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

10/3/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16536022 BIOSIS NO.: 200200129533
Functional study of TCR-dependent T cell activation in defective T lymphocytes of PNH patients
AUTHOR: Alfinito Fiorella (Reprint); Ruggiero Giuseppina; Andretta Claudia (Reprint); Terrazzano Giuseppe; Zappacosta Serafino; Rotoli Bruno (Reprint)
AUTHOR ADDRESS: Hematology, University of Naples Federico II, Naples, Italy **Italy
JOURNAL: Blood 98 (11 Part 1): p25a November 16, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001; 20011207
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

10/3/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16456977 BIOSIS NO.: 200200050488
Detection of mutation in a CD40 ligand gene
AUTHOR: Spriggs M K; Armitage R J; Fanslow W C III
AUTHOR ADDRESS: Seattle, Wash., USA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1191 (3): p1909 Oct. 15, 1996 ***1996***
MEDIUM: print
PATENT NUMBER: US 5565321 PATENT DATE GRANTED: Oct. 15, 1996 19961015
PATENT CLASSIFICATION: 435-6 PATENT ASSIGNEE: IMMUNEX CORPORATION
PATENT COUNTRY: USA
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Citation
LANGUAGE: English

10/3/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16413107 BIOSIS NO.: 200200006618
Predictive value of the detection of CD40 and Cytotoxic T-Lymphocytes in early rejecting renal allografts
AUTHOR: Mengel M (Reprint); Mueller I (Reprint); Behrend M; von Woellwarth J; Kreipe H (Reprint)
AUTHOR ADDRESS: Institut fuer Pathologie der Medizinischen Hochschule, Hannover, Germany**Germany
JOURNAL: Kidney and Blood Pressure Research 24 (4-6): p272-273 2001 2001
MEDIUM: print
CONFERENCE/MEETING: Joint Scientific Meeting of the Nephrology Society and the German Working Group for Clinical Nephrology Munster, Germany September 29-October 02, 2001; 20010929
ISSN: 1420-4096
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

10/3/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16399969 BIOSIS NO.: 200100571808
Maturation of dendritic cells from ovarian cancer patients
AUTHOR: Zavadova Eva; Savary Cherylyn A; Templin Stacie; Verschraegen Claire F; Freedman Ralph S (Reprint)
AUTHOR ADDRESS: Department of Gynecologic Oncology, University of Texas M. D. Anderson Cancer Center, 1515 Holcombe Boulevard, Houston, TX, 77030, USA**USA
JOURNAL: Cancer Chemotherapy and Pharmacology 48 (4): p289-296 October, 2001 2001
MEDIUM: print
ISSN: 0344-5704
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16362853 BIOSIS NO.: 200100534692
Photochemically enhanced binding of small molecules to the tumor necrosis factor receptor-1 inhibits the binding of TNF-alpha
AUTHOR: Carter Percy H (Reprint); Scherle Peggy A; Muckelbauer Jodi A; Voss Matthew E; Liu Rui-Qin; Thompson Lorin A; Tebben Andrew J; Solomon Kimberly A; Lo Yvonne C; Li Zhong; Strzemienski Paul; Yang Gengjie; Falahatpisheh Nikoo; Xu Meizhong; Wu Zhongren; Farrow Neil A; Ramnarayan Kal; Wang Jing; Rideout Darryl; Yalamoori Venkatachalapathi; Domaille Peter; Underwood Dennis J; Trzaskos James M; Friedman Steven M; Newton Robert C; Decicco Carl P
AUTHOR ADDRESS: Experimental Station, DuPont Pharmaceuticals Company, Route 141 and Henry Clay Road, Wilmington, DE, 19880-0500, USA**USA

JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 98 (21): p11879-11884 October 9, 2001 2001
MEDIUM: print
ISSN: 0027-8424
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16352941 BIOSIS NO.: 200100524780
NMDA receptor subtype activation by macrophage secretory products: A pathway for neural compromise in HIV-1 associated dementia
AUTHOR: Xiong H (Reprint); McCabe L (Reprint); Skifter D; Monaghan D T; Zheng J (Reprint); Gendelman H E (Reprint)
AUTHOR ADDRESS: Ctr Neurovirol and Neurodegenerative Disorders, Univ NE Med Ctr, Omaha, NE, USA**USA
JOURNAL: Society for Neuroscience Abstracts 27 (1): p1272 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 31st Annual Meeting of the Society for Neuroscience San Diego, California, USA November 10-15, 2001; 20011110
ISSN: 0190-5295
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

10/3/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16291755 BIOSIS NO.: 200100463594
Protective effect of human CD40-Ig fusion protein in a murine model of acute graft-versus-host disease
AUTHOR: Liu Hezhong; Mao Ning; Hou Chunmei; Li Xiusen; Shen Beifen; Tang Pei-Hsien (Reprint)
AUTHOR ADDRESS: Institute of Basic Medical Sciences, Academy of Military Medical Sciences, Beijing, 100850, China**China
JOURNAL: Chinese Medical Journal (English Edition) 114 (7): p685-689 July, 2001 2001
MEDIUM: print
ISSN: 0366-6999
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/11 (Item 11 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16257315 BIOSIS NO.: 200100429154
Costimulatory molecules in the developing human gastrointestinal tract: A pathway for fetal allergen priming
AUTHOR: Jones Catherine A (Reprint); Vance Gillian H S; Power Lynsey L; Pender Sylvia L F; MacDonald Thomas T; Wamer John O
AUTHOR ADDRESS: Child Health (803), Southampton General Hospital, Southampton, SO16 6YD, UK**UK

JOURNAL: Journal of Allergy and Clinical Immunology 108 (2): p235-241 August, 2001 2001
MEDIUM: print
ISSN: 0091-6749
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/12 (Item 12 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16255232 BIOSIS NO.: 200100427071
Nucleic acid encoding CD40 associated proteins
AUTHOR: Reed John C; Sato Takaaki
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1248 (4): July 24, 2001 2001
MEDIUM: e-file
PATENT NUMBER: US 6265556 PATENT DATE GRANTED: July 24, 2001 20010724
PATENT CLASSIFICATION: 536-231 PATENT ASSIGNEE: The Burnham Institute
PATENT COUNTRY: USA
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

10/3/13 (Item 13 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16214018 BIOSIS NO.: 200100385857
Secretion of anti-citrulline-containing peptide antibody by B lymphocytes in rheumatoid arthritis
AUTHOR: Reparon-Schuijt Carelle C; van Esch Wim J E; van Kooten Cees; Schellekens Gerard A; de Jong Ben A W; van Venrooij Walther J; Breedveld Ferdinand C; Verweij Cornelis L (Reprint)
AUTHOR ADDRESS: Department of Rheumatology, Leiden University Medical Center, C4-R2, 2300 RC, Leiden, Netherlands**Netherlands
JOURNAL: Arthritis and Rheumatism 44 (1): p41-47 January, 2001 2001
MEDIUM: print
ISSN: 0004-3591
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/14 (Item 14 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16203390 BIOSIS NO.: 200100375229
Mechanisms of CD23 hyperexpression on B cells from patients with rheumatoid arthritis
AUTHOR: De Miguel Sonia; Galocha Begona; Jover Juan A; Banares Antonio; Hernandez-Garcia Cesar; Garcia-Asenjo Jose A; Fernandez-Gutierrez Benjamin (Reprint)
AUTHOR ADDRESS: Service of Rheumatology, Hospital Clinico San Carlos, 28040, Madrid, Spain**Spain
JOURNAL: Journal of Rheumatology 28 (6): p1222-1228 June, 2001 2001

MEDIUM: print
ISSN: 0315-162X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/15 (Item 15 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16165475 BIOSIS NO.: 200100337314
CD40 ligand-deficient mice are protected against cerulein-induced acute pancreatitis and pancreatitis-associated lung injury
AUTHOR: Frossard Jean Louis (Reprint); Kwak Brenda; Chanson Marc; Morel Philippe; Hadengue Antoine; Mach Francois
AUTHOR ADDRESS: Division of Gastroenterology, Geneva University Hospitals, Rue Micheli du Crest, 1211, Geneva 14, Switzerland**Switzerland
JOURNAL: Gastroenterology 121 (1): p184-194 July, 2001 2001
MEDIUM: print
ISSN: 0016-5085
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/16 (Item 16 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16165195 BIOSIS NO.: 200100337034
Varicella-zoster virus infection of human dendritic cells and transmission to T cells: Implications for virus dissemination in the host
AUTHOR: Abendroth Allison (Reprint); Morrow Gavin; Cunningham Anthony L; Slobedman Barry
AUTHOR ADDRESS: Centre for Virus Research, Westmead Millennium Institute, Westmead Hospital, Rm. 3024, Westmead, NSW, 2145, Australia**Australia
JOURNAL: Journal of Virology 75 (13): p6183-6192 July, 2001 2001
MEDIUM: print
ISSN: 0022-538X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/17 (Item 17 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16163944 BIOSIS NO.: 200100335783
Following direct CD40 activation, human primary microglial cells produce IL-12 p40 but not bioactive IL-12 p70
AUTHOR: de Herve M G de Goer; Delfraissy J F; Taoufik Y (Reprint)
AUTHOR ADDRESS: Laboratoire d'Immunologie, Service d'Hematologie, Hopital de Bicetre, 78 Rue du General Leclerc, Le Kremlin-Bicetre Cedex, 94270, France**France
JOURNAL: Cytokine 14 (2): p88-96 21 April, 2001 2001
MEDIUM: print
ISSN: 1043-4666
DOCUMENT TYPE: Article
RECORD TYPE: Abstract

LANGUAGE: English

10/3/18 (Item 18 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16153110 BIOSIS NO.: 200100324949
A novel system for convenient detection of low-affinity receptor-ligand interactions: Chelator-lipid liposomes engrafted with recombinant CD4 bind to cells expressing MHC class II
AUTHOR: Van Broekhoven Christina L; Altin Joseph G (Reprint)
AUTHOR ADDRESS: School of Biochemistry and Molecular Biology, Faculty of Science, Australian National University, Canberra, ACT, 0200, Australia** Australia
JOURNAL: Immunology and Cell Biology 79 (3): p274-284 June, 2001 2001
MEDIUM: print
ISSN: 0818-9641
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/19 (Item 19 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16150911 BIOSIS NO.: 200100322750
Generation of CD8+ T cell lines with specific cytotoxicity for autologous Chronic Lymphocytic Leukemia B cells
AUTHOR: Deforce Dieter L (Reprint); Chu Peter (Reprint); Rassenti Laura Z (Reprint); Mendoza Robert (Reprint); Raz Eyal (Reprint); Kipps Thomas J (Reprint)
AUTHOR ADDRESS: Hematology/Oncology, UCSD School of Medicine, La Jolla, CA, USA**USA
JOURNAL: Blood 96 (11 Part 1): p368a November 16, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000; 20001201
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

10/3/20 (Item 20 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16139863 BIOSIS NO.: 200100311702
Tumor-reactive T-cell lines can be generated in the presence of large numbers of CLL cells: Implications for vaccine development in CLL
AUTHOR: Iakhnina Elena (Reprint); Spaner David (Reprint); Lorenzo Maria N (Reprint); Buckstein Rena (Reprint); Imrie Kevin (Reprint); Reis Marciano (Reprint); Berinstein Neil L (Reprint)
AUTHOR ADDRESS: Advanced Therapeutics Program, Toronto Sunnybrook Regional Cancer Centre, Sunnybrook and Women's College Health Sciences Centre, Toronto, ON, Canada**Canada
JOURNAL: Blood 96 (11 Part 1): p162a November 16, 2000 2000

MEDIUM: print
CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000; 20001201
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DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

10/3/21 (Item 21 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16139813 BIOSIS NO.: 200100311652
Ku86v translocates into cell membrane and associates with Ku70 in human multiple myeloma (MM) cells
AUTHOR: Tai YuTzu (Reprint); Podar Klaus (Reprint); Hideshima Teru (Reprint); Wang FengFei (Reprint); Davies Faith E (Reprint); Young Gloria (Reprint); Chauhan Dharminder (Reprint); Lin Boris (Reprint); Treon Steve P (Reprint); Mitsiades Constantine S (Reprint); Lentzsch Suzanne (Reprint); Gupta Deepak (Reprint); Hayashi Toshiaki (Reprint); Mitsiades Nicholas (Reprint); Raje Noopur (Reprint); Koike Manaba; Taccioli Guillermo E; Anderson Kenneth C (Reprint)
AUTHOR ADDRESS: Adult Oncology, Dana-Farber Cancer Institute, Boston, MA, USA**USA
JOURNAL: Blood 96 (11 Part 1): p158a November 16, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000; 20001201
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

10/3/22 (Item 22 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16139697 BIOSIS NO.: 200100311536
The role of 4-1BB ligand in CD8+ T cell responses in vitro
AUTHOR: Galy Anne (Reprint); Laderach Diego (Reprint)
AUTHOR ADDRESS: Barbara Ann Karmanos Cancer Institute, Wayne State University, Detroit, MI, USA**USA
JOURNAL: Blood 96 (11 Part 1): p240a-241a November 16, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000; 20001201
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ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

10/3/23 (Item 23 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16139472 BIOSIS NO.: 200100311311
The proto-oncogene c-Src is expressed in normal and transformed B lymphocytes
AUTHOR: Bernard Frederic (Reprint); Fischer Alain; Hivroz Claire
AUTHOR ADDRESS: Hemato Oncologie Peddiatrie, Hopital Arnaud de Villeneuve, Montpellier, France**France
JOURNAL: Blood 96 (11 Part 2): p39b November 16, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000; 20001201
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RECORD TYPE: Abstract
LANGUAGE: English

10/3/24 (Item 24 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16131232 BIOSIS NO.: 200100303071
Identification of a leukemic counterpart of the "plasmacytoid dendritic cells"
AUTHOR: Chaperot Laurence (Reprint); Bendriss Nathalie; Gressin Remy; Bensa Jean-Claude (Reprint); Sotto Jean-Jacques; Briere Francine; Plumas Joel (Reprint); Jacob Marie-Christine (Reprint)
AUTHOR ADDRESS: Research Group on Lymphoma, Research and Development, EFS Rhone Alpes, Grenoble, France**France
JOURNAL: Blood 96 (11 Part 1): p316a November 16, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000; 20001201
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

10/3/25 (Item 25 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16127689 BIOSIS NO.: 200100299528
Functional soluble CD100/Sema4D released from activated lymphocytes: Possible role in normal and pathologic immune responses
AUTHOR: Wang Xiaosong; Kumanogoh Atsushi; Watanabe Chie; Shi Wei; Yoshida Kanji; Kikutani Hitoshi (Reprint)
AUTHOR ADDRESS: Department of Molecular Immunology, Research Institute for Microbial Diseases, Osaka University, 3-1 Yamada-oka, Suita, Osaka, 565-0871, Japan**Japan
JOURNAL: Blood 97 (11): p3498-3504 June 1, 2001 2001
MEDIUM: print
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/26 (Item 26 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

16127610 BIOSIS NO.: 200100299449
CD154 (CD40-ligand) gene therapy for chronic lymphocytic leukemia leads to in vivo activation of the STAT1 signaling pathway
AUTHOR: Battle Traci E (Reprint); Wierda William G; Rassenti Laura Z; Kipps Thomas J; Frank David A (Reprint)
AUTHOR ADDRESS: Dept. of Adult Oncology, Dana-Farber Cancer Institute, Boston, MA, USA**USA
JOURNAL: Blood 96 (11 Part 1): p157a November 16, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000; 20001201
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

10/3/27 (Item 27 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16121664 BIOSIS NO.: 200100293503
T cell responses in CLL: Superiority of Th2 responses might contribute to reduced cytotoxic autologous antitumor responses
AUTHOR: Krackhardt Angela M (Reprint); Harig Sabine (Reprint); Witzens Mathias (Reprint); Barrett Patrick (Reprint); Broderick Ryan (Reprint); Gribben John G (Reprint)
AUTHOR ADDRESS: Adult Oncology, Dana Farber Cancer Institute, Boston, MA, USA**USA
JOURNAL: Blood 96 (11 Part 1): p28a November 16, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000; 20001201
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

10/3/28 (Item 28 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16119561 BIOSIS NO.: 200100291400
Specific blockade by CD54 and MHC II of CD40-mediated signaling for B cell proliferation and survival
AUTHOR: Doyle Iris S; Hollmann C Annette; Crispe I Nicholas; Owens Trevor (Reprint)
AUTHOR ADDRESS: Neuroimmunology, Montreal Neurological Institute, 3801 University Street, Montreal, PQ, H3A 2B4, Canada**Canada
JOURNAL: Experimental Cell Research 265 (2): p312-318 May 1, 2001 2001
MEDIUM: print
ISSN: 0014-4827
DOCUMENT TYPE: Article
RECORD TYPE: Abstract

LANGUAGE: English

10/3/29 (Item 29 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16119357 BIOSIS NO.: 200100291196
CpG DNA promotes the maturation and function of human monocyte-derived dendritic cells (MoDC) via indirect effects
AUTHOR: Chan Anissa S H (Reprint); Pond David P (Reprint); Panoskaltsis-Mortari Angela (Reprint); Wang Jianli (Reprint); Krieg Arthur M; Blazar Bruce R (Reprint); Chen Wei (Reprint)
AUTHOR ADDRESS: Univ of Minn Cancer Center, Minneapolis, MN, USA**USA
JOURNAL: Blood 96 (11 Part 2): p210b November 16, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000; 20001201
SPONSOR: American Society of Hematology
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DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

10/3/30 (Item 30 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16110760 BIOSIS NO.: 200100282599
Expression of CD23/CD21 and CD40/CD40 ligand in vernal keratoconjunctivitis
AUTHOR: Abu El-Asrar Ahmed M (Reprint); Fatani Rashed A; Missotten Luc; Geboes Karel
AUTHOR ADDRESS: Department of Ophthalmology, King Abdulaziz University Hospital, Airport Road, Riyadh, 11411, Saudi Arabia**Saudi Arabia
JOURNAL: Eye (London) 15 (2): p217-224 April, 2001 2001
MEDIUM: print
ISSN: 0950-222X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/31 (Item 31 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16104897 BIOSIS NO.: 200100276736
CD40 signaling in B cells regulates the expression of pim-1 via the NF-kappaB pathway
AUTHOR: Zhu Mindy (Reprint); Ramirez Luis; Lee Rosaline (Reprint); Pelech Steve; Bishop Gail; Gold Michael (Reprint)
AUTHOR ADDRESS: Department of Microbiology and Immunology, University of British Columbia, Vancouver, B.C., V6T 1Z3, Canada**Canada
JOURNAL: FASEB Journal 15 (4): pA703 March 7, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001; 20010331
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract

RECORD TYPE: Abstract
LANGUAGE: English

10/3/32 (Item 32 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16095914 BIOSIS NO.: 200100267753
In vitro effects of retinoic acid on differentiation of murine dendritic cells from bone marrow stem cells
AUTHOR: Hites Greg William (Reprint); Hoag Kathleen A (Reprint)
AUTHOR ADDRESS: Slippery Rock University of Pennsylvania, 123 Vincent Science Hall, Slippery Rock, PA, 16057, USA**USA
JOURNAL: FASEB Journal 15 (4): pA672 March 7, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001; 20010331
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

10/3/33 (Item 33 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16074224 BIOSIS NO.: 200100246063
Localization of recombination activating gene 1/green fluorescent protein (RAG1/GFP) expression in secondary lymphoid organs after immunization with T-dependent antigens in rag1/gfp knockin mice
AUTHOR: Igarashi Hideya; Kuwata Naomi; Kiyota Kumiko; Sumita Kiminobu; Suda Toshio; Ono Shiro; Bauer Steven R; Sakaguchi Nobuo (Reprint)
AUTHOR ADDRESS: Department of Immunology, Kumamoto University School of Medicine, 2-2-1, Honjo, Kumamoto, 860-0811, Japan**Japan
JOURNAL: Blood 97 (9): p2680-2687 May 1, 2001 2001
MEDIUM: print
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/34 (Item 34 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16071417 BIOSIS NO.: 200100243256
Survivin is expressed on CD40 stimulation and interfaces proliferation and apoptosis in B-cell chronic lymphocytic leukemia
AUTHOR: Granziero Luisa; Ghia Paolo; Circosta Paola; Gottardi Daniela; Strola Giuliana; Geuna Massimo; Montagna Licia; Piccoli Paola; Chilosi Marco; Caligaris-Cappio Federico (Reprint)
AUTHOR ADDRESS: University Division of Clinical Immunology and Hematology, Ospedale Mauriziano Umbeirto I, Largo Turati 62, 10128, Torino, Italy** Italy
JOURNAL: Blood 97 (9): p2777-2783 May 1, 2001 2001
MEDIUM: print
ISSN: 0006-4971

DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/35 (Item 35 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16061511 BIOSIS NO.: 200100233350
Study of expression and purification of CD40-Ig fusion protein in CHO cells
AUTHOR: Liu Hezhong (Reprint); Mao Ning; Hou Chunmei
AUTHOR ADDRESS: Institute of Basic Medical Sciences, Academy of Military Medical Sciences, Beijing, 100850, China**China
JOURNAL: Zhonghua Weishengwuxue He Mianyixue Zazhi 21 (2): p151-155 March, 2001 2001
MEDIUM: print
ISSN: 0254-5101
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: Chinese

10/3/36 (Item 36 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16039052 BIOSIS NO.: 200100210891
CD40 is not detected on human prostate cancer cells by immunohistologic techniques
AUTHOR: Moghaddami Mahin; Cohen Penny; Stapleton Alan M F; Brown Michael P (Reprint)
AUTHOR ADDRESS: Department of Medical Oncology, Royal Adelaide Hospital, North Terrace, Level 7, East Wing, Adelaide, SA, 5000, Australia** Australia
JOURNAL: Urology 57 (3): p573-578 March, 2001 2001
MEDIUM: print
ISSN: 0090-4295
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/37 (Item 37 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16016171 BIOSIS NO.: 200100188010
CD40 is expressed on human peritoneal mesothelial cells and upregulates the production of interleukin-15 and RANTES
AUTHOR: Basok Anna; Shnaider Alla; Man Limor; Chaimovitz Cidio; Douvdevani Amos (Reprint)
AUTHOR ADDRESS: Nephrology Laboratory, Soroka Medical Center, Beer-Sheva, 84101, Israel**Israel
JOURNAL: Journal of the American Society of Nephrology 12 (4): p695-702 April, 2001 2001
MEDIUM: print
ISSN: 1046-6673
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/38 (Item 38 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16013452 BIOSIS NO.: 200100185291
Treatment of individuals exhibiting defective CD40L
AUTHOR: Spriggs Melanie K; Armitage Richard J; Fanslow William C; Widmer Michael B (Reprint)
AUTHOR ADDRESS: Seattle, WA, USA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1237 (4): Aug. 22, 2000 ***2000***
MEDIUM: e-file
PATENT NUMBER: US 6106832 PATENT DATE GRANTED: August 22, 2000 20000822
PATENT CLASSIFICATION: 424-1341 PATENT ASSIGNEE: Immunex Corporation
PATENT COUNTRY: USA
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

10/3/39 (Item 39 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15969862 BIOSIS NO.: 200100141701
Influence of costimulatory molecules on immune response to Leishmania major by human cells in vitro
AUTHOR: Brodskyn Claudia I; DeKrey Gregory K; Titus Richard G (Reprint)
AUTHOR ADDRESS: Department of Pathology, College of Veterinary Medicine and Biomedical Sciences, Colorado State University, Fort Collins, CO, 80523, USA**USA
JOURNAL: Infection and Immunity 69 (2): p665-672 February, 2001 2001
MEDIUM: print
ISSN: 0019-9567
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/40 (Item 40 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15956615 BIOSIS NO.: 200100128454
Analysis of antigen presenting cell derived exosomes, based on immuno-magnetic isolation and flow cytometry
AUTHOR: Clayton Aled (Reprint); Court Jacquelyn; Navabi Hossein; Adams Malcolm; Mason Malcolm D; Hobot Jan A; Newman Geoff R; Jasani Bharat
AUTHOR ADDRESS: Section of Clinical Oncology, Velindre Hospital, University of Wales College of Medicine, Whitchurch, Cardiff, CF14 2TL, UK**UK
JOURNAL: Journal of Immunological Methods 247 (1-2): p163-174 1 January, 2001 2001
MEDIUM: print
ISSN: 0022-1759
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/41 (Item 41 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15931550 BIOSIS NO.: 200100103389
CD40 expression in uterine tissues: A key regulator of cytokine expression by fibroblasts
AUTHOR: King Anne E (Reprint); Kelly Rodney W; Critchley Hilary O D; Malmstrom Anders; Sennstrom Maria; Phipps Richard P
AUTHOR ADDRESS: Medical Research Council Reproductive Biology Unit, Center for Reproductive Biology, University of Edinburgh, Edinburgh, EH3 9ET, UK **UK
JOURNAL: Journal of Clinical Endocrinology and Metabolism 86 (1): p405-412 January, 2001 2001
MEDIUM: print
ISSN: 0021-972X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/42 (Item 42 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15923642 BIOSIS NO.: 200100095481
Characterization of cytokine, growth factor receptor, costimulatory and adhesion molecule expression patterns of bone marrow blasts in relapsed childhood B cell precursor all
AUTHOR: Kebelmann-Betzing Christian (Reprint); Koerner Gabriele; Badiali Lucia; Buchwald Dirk; Moericke Anja; Korte Alexander; Koechling Joachim; Wu Shuling; Kappelmeier Diane; Oettel Klaus; Henze Guenter; Seeger Karlheinz
AUTHOR ADDRESS: Department of Pediatric Oncology/Hematology, Charite, Humboldt-University at Berlin, Augustenburger Platz 1, Mail drop, Germany **Germany
JOURNAL: Cytokine 13 (1): p39-50 7 January, 2001 2001
MEDIUM: print
ISSN: 1043-4666
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/43 (Item 43 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15890263 BIOSIS NO.: 200100062102
Role for CD40-CD40 ligand interactions in the immune response to solid tumours
AUTHOR: Alexandroff Anton B; Jackson Andrew M; Paterson T; Haley Joanne L; Ross James A; Longo D L; Murphy W J; James Keith (Reprint); Taub Dennis D
AUTHOR ADDRESS: Department of Clinical and Surgical Sciences, Lister Laboratories, Royal Infirmary, Edinburgh University, Lauriston Place, Edinburgh, EH3 9YW, UK**UK
JOURNAL: Molecular Immunology 37 (9): p515-526 June, 2000 2000
MEDIUM: print
ISSN: 0161-5890
DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: English

10/3/44 (Item 44 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15878803 BIOSIS NO.: 200100050642
Long-term study of a female hyper-IgM immunodeficiency
AUTHOR: Kaneko Hideo (Reprint); Fukao Toshiyuki; Inoue Ryousuke; Kasahara Kimiko; Matsui Eiko; Kondo Naomi
AUTHOR ADDRESS: Department of Pediatrics, Gifu University School of Medicine, Tukasa-machi 40, Gifu, 500-8706, Japan**Japan
JOURNAL: Experimental and Clinical Immunogenetics 17 (4): p173-178 November, 2000 2000
MEDIUM: print
ISSN: 0254-9670
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/45 (Item 45 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15878266 BIOSIS NO.: 200100050105
Antigen-independent appearance of recombination activating gene (RAG)-positive bone marrow B cells in the spleens of immunized mice
AUTHOR: Gartner Frank; Alt Frederick W (Reprint); Monroe Robert J; Seidl Katherine J
AUTHOR ADDRESS: Howard Hughes Medical Institute, Children's Hospital, Enders 861, 300 Longwood Ave., Boston, MA, 02115, USA**USA
JOURNAL: Journal of Experimental Medicine 192 (12): p1745-1754 December 18, 2000 2000
MEDIUM: print
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/46 (Item 46 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15812229 BIOSIS NO.: 200000530542
Role of platelet P-selectin and CD40 ligand in the induction of monocytic tissue factor expression
AUTHOR: Lindmark Eva; Tenno Taavo; Siegbahn Agneta (Reprint)
AUTHOR ADDRESS: Department of Medical Sciences, Clinical Chemistry, University Hospital, S-75185, Uppsala, Sweden**Sweden
JOURNAL: Arteriosclerosis Thrombosis and Vascular Biology 20 (10): p 2322-2328 October, 2000 2000
MEDIUM: print
ISSN: 1079-5642
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/47 (Item 47 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15773902 BIOSIS NO.: 200000492215
Signals sustaining human immunoglobulin V gene hypermutation in isolated germinal centre B cells
AUTHOR: Dahlenborg K; Pound J D; Gordon J; Borrebaeck C A K; Carlsson R (Reprint)
AUTHOR ADDRESS: BioInvent Therapeutic AB, S-223 70, Lund, Sweden**Sweden
JOURNAL: Immunology 101 (2): p210-217 October, 2000 2000
MEDIUM: print
ISSN: 0019-2805
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/48 (Item 48 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15768276 BIOSIS NO.: 200000486589
Role and expression of CD4O on human retinal pigment epitlielial cells
AUTHOR: Willermain Francois (Reprint); Caspers-Velu Laure; Baudson Nathalie ; Dubois Christine; Hamdane Malika; Willems Fabienne; Velu Thierry; Bruyns Catherine
AUTHOR ADDRESS: ULB, Cmpus Erasme, I.R.I.B.H.N., Route de Lennik 808, Building C, 6th Floor, Room C6 117, Bruxelles, Belgium**Belgium
JOURNAL: IOVS 41 (11): p3485-3491 OCTOBER, 2000 2000
MEDIUM: print
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/49 (Item 49 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15738808 BIOSIS NO.: 200000457121
Hypereosinophilic syndrome presenting as cutaneous necrotizing eosinophilic vasculitis and Raynaud's phenomenon complicated by digital gangrene
AUTHOR: Jang K-A (Reprint); Lim Y-S; Choi J-H; Sung K-J; Moon K-C; Koh J-K
AUTHOR ADDRESS: Department of Dermatology, College of Medicine, Paik Hospital, Inje-University, 85, 2-Ga, Jur-Dong, Chung-Ku, Seoul, 100-032, South Korea**South Korea
JOURNAL: British Journal of Dermatology 143 (3): p641-644 September, 2000 2000
MEDIUM: print
ISSN: 0007-0963
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/50 (Item 50 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15735618   BIOSIS NO.: 200000453931
Induction of IgE synthesis by genetically modified CD8+ T cells of a patient with adenosine deaminase deficiency
AUTHOR: Yanagihara Yukiyoshi (Reprint); Kajiwara Keiichi; Basaki Yuji; Ikizawa Koichi; Mori Miyuki; Akiyama Kazuo; Kawamura Nobuaki; Sakiyama Yukio
AUTHOR ADDRESS: Clinical Research Center for Allergy, National Sagamihara Hospital, 18-1 Sakuradai, Sagamihara, 228-8522, Japan**Japan
JOURNAL: Allergology International 49 (3): p195-204 September, 2000
2000
MEDIUM: print
ISSN: 1323-8930
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
? t s10/7/1-6,30

 10/7/1   (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)

16963642   BIOSIS NO.: 200200557153
Characterization of soluble CD40 ligand released from human activated platelets
AUTHOR: Jin Yinzhu; Nonoyama Shigeaki (Reprint); Morio Tomohiro; Imai Kohsuke; Ochs Hans D; Mizutani Shuki
AUTHOR ADDRESS: Division of Human Ontogeny and Childhood Development, Graduate School, Tokyo Medical and Dental University, 1-5-45, Yushima, Bunkyo-ku, Tokyo, 113-8519, Japan**Japan
JOURNAL: Journal of Medical and Dental Sciences 48 (1): p23-27 Mar., 2001
2001
MEDIUM: print
ISSN: 1342-8810
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We report here that soluble CD40 ligand (sCD40L) is released from human platelets when activated with collagen or thrombin. The sCD40L was detectable in the culture supernatants of platelets within 30 min after stimulation in vitro, and reached maximal levels in 3 h. The release was blocked by the metalloproteinase inhibitor, KB8301, indicating that the soluble CD40L is made by cleaving the membrane bound CD40L expressed on activated platelets. The sCD40L was undetectable in the supernatant of the activated platelets obtained from patients with X-linked hyper IgM syndrome (XHIM), who have defects in CD40L gene. Since sCD40L has been shown to have biologic function on the activation of vascular endothelial cells and B cells, these findings suggest that platelets play some roles in both inflammation and humoral immune response by releasing soluble CD40L.

 10/7/2   (Item 2 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)

16616423   BIOSIS NO.: 200200209934
Direct effects of CD40L on CD40+AML blasts: Proliferation, self renewal, rescue from apoptosis and production of cytokines
AUTHOR: Aldinucci Donatella (Reprint); Poletto Dalisa (Reprint); Nanni Paola (Reprint); Degan Massimo (Reprint); Rupolo Maurizio (Reprint);

Pinto Antonio (Reprint); Gattei Valter (Reprint)
AUTHOR ADDRESS: Nucleo di Ricerca Clinica e Laboratoristica in Ematologia, Centro di Riferimento Oncologico, IRCCS, Aviano, PN, Italy**Italy
JOURNAL: Blood 98 (11 Part 1): p589a November 16, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001; 20011207
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: CD40 is a co-stimulatory molecule belonging to the TNF-R superfamily playing a key role in the activation of professional antigen presenting cells (APC) during the immune response. The expression of CD40 by several hematological malignancies, mainly of B-cell origin, suggested the use of CD40 triggering to overcome the immunosurveillance escape of neoplastic cells. Indeed, data so far produced focused almost exclusively on the role of CD40 triggering as a tool to activate the APC function of tumor cells, while the direct effects of CD40 engagement have not been yet investigated in detail. In the present study, we wish to clarify the consequence of CD40 ligation in terms of activation, proliferation, apoptosis and cytokines production in ***CD40*** -expressing AML blasts. In a series of 67 AMLs samples encompassing all the FAB phenotypes, we demonstrated CD40 expression in 25/67 cases (37%), the highest frequency being ***detected*** among the monocytic subtypes (11/13, 84%). The expression of CD40 was up-regulated or de novo induced upon exposure of blasts cells to several cytokines including IL-1-alpha, IL-3, IL-4, GM-CSF, IFN-gamma and TNF-alpha. Conversely, the expression of the ***CD40*** counter-receptor ***CD40L*** was never ***detected*** on AML blasts. Exposure of AML blasts to different concentrations of soluble (s) CD40L resulted in a dose-dependent enhancement of their proliferative response, as determined by radiolabeled thymidine uptake (stimulation index ranging from 1.7 to 9.3). Consistently, sCD40L was able to induce an increase in the number of AML blast colonies (fold increase ranging from 2.9 to 16) and of the self-renewal capacity of AML blasts, as assessed by clonogenic growth after replating in semisolid medium. CD40 engagement up-regulated cells surface expression of the adhesion molecules CD54, CD58, and CD15 and resulted in a CD54-dependent homotypic aggregation of the leukemic cells. Moreover, sCD40L inhibited early apoptotic events in AML blasts cultured in the absence of serum, as demonstrated by a decrease, evaluated in flow cytometry, of the percentage of APO2.7 (31% vs. 47% in controls) and Annexin-V (29% vs. 48% in controls) positive cells. Consistently, co-cultures of AML blasts in the presence of CD40L-transfected fibroblasts resulted in an increased expression of the anti-apoptotic protein Bcl-xL, as assessed by Western blotting, while no effect on Bax and Bcl-2 expression was observed. Finally, CD40 triggering determined an increase of GM-CSF and/or IL-6 production in 9/12 (75%) AML samples (fold increase ranging from 1.34 to 12.4 or 3.5 to 350, respectively). Our data demonstrated that CD40 engagement on AML had a direct effect on proliferation, rescue from apoptosis and production of cytokines involved in AML growth. This notion suggests a caution in the wide therapeutic use of CD40L with the aim to restore deficient APC function of tumor cells by CD40 triggering. A rationale for a clinical use of CD40L in AMLs might be more likely to potentiate the antileukemic effects of specific chemotherapic agents. Alternatively, CD40 might be viewed as an additional target molecule for antibody therapies.

10/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16558296 BIOSIS NO.: 200200151807
CD40 ligand accumulation during storage of human platelet concentrates: Implications for transfusion complications
AUTHOR: Vanderlinde Elizabeth (Reprint); Kaufman Julia; Phipps Richard; Blumberg Neil (Reprint)
AUTHOR ADDRESS: Transfusion Medicine, Univ. of Rochester Medical Center, Rochester, NY, USA**USA
JOURNAL: Blood 98 (11 Part 2): p112b November 16, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 2 Orlando, Florida, USA December 07-11, 2001; 20011207
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: INTRODUCTION: CD40 ligand (CD40L, CD154), via interaction with its receptor CD40, induces production of several pro-inflammatory cytokines and mediators including IL-1, IL-6, IL-8, the cyclo-oxygenase-2 enzyme and prostaglandin E2. Prostaglandin E2 is the main fever inducer in humans. Traditionally, ***CD40L*** expressed by activated T cells engaged CD40 on macrophages or dendritic cells to induce production of cytokine mediators. However, platelets have emerged as a potent source of preformed ***CD40L*** capable of immune cell activation. Recently, high concentrations of bioactive CD40L were detected in leukoreduced human platelet concentrates during the 5 day storage period, and greater than 50% of stored platelets expressed CD40L on their surface membrane (Lancet 357: 2023, 2001). Soluble CD40L (sCD40L) is thus a candidate mediator for febrile transfusion reactions, TRALI (transfusion related acute lung injury) and transfusion immunomodulation. To further investigate CD40L's presence in human platelet concentrates we studied the kinetics of platelet secretion of CD40L into the plasma supernatant and the expression of CD40L on the platelet surface membrane during storage. We also investigated the effects of thrombin stimulation on secretion and membrane expression of CD40L. METHODS: Human platelet concentrates were obtained from either the regional American Red Cross Blood Services or prepared by the platelet rich plasma method from donated whole blood in our institution's blood bank. Soluble CD40L was measured by a specific ELISA and membrane-bound CD40L was assessed by flow cytometry using an anti-CD40L monoclonal antibody and appropriate controls. RESULTS: Thrombin stimulation of platelets induces secretion of CD40L, in vitro, with increased amounts of sCD40L in platelet preparations over time of exposure. In one experiment measuring sCD40L from thrombin-stimulated platelets, at 15 minutes after exposure to thrombin, sCD40L concentration was 2.4 ng/ml versus 0.8 ng/ml in the unstimulated control. At three hours after exposure, sCD40L was 5.4 ng/ml versus 1.5 ng/ml in the control. CD40L was also elevated in apheresis platelet concentrates on day three of storage (16.0 ng/ml mean concentration; 45% of platelets positive for surface expression), and there was little change as the product aged to its outdate. Random donor platelet concentrates at six hours of storage (n=2) contained 5.9 ng/ml and 3.2 ng/ml sCD40L; 67% and 38% of platelets were positive for membrane-associated CD40L, respectively. Peak concentrations of sCD40L at 78 hours and 123 hours were 34.0 ng/ml and 23.9 ng/ml, respectively, with platelets positive for CD40L membrane expression ranging from 43% to 78%

and 24% to 53%, respectively, over days one through five of storage. Normally, the level of sCD40L in human plasma from healthy subjects is undetectable and unstimulated platelets do not express CD40L. Levels of sCD40L seen in stored platelet concentrates are only seen in vivo in patients with florid inflammatory states. CONCLUSION: CD40L secretion and membrane expression by platelets increases within hours after preparation of both apheresis and random donor platelet concentrates. The platelet agonist thrombin induces increased secretion and expression of CD40L from platelets, in vitro. Future studies of soluble and membrane-bound forms of CD40L infused with platelet concentrates are needed to determine the relationship between CD40L infusion and the occurrence of febrile transfusion reactions, TRALI and transfusion immunomodulation.

10/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16536022 BIOSIS NO.: 200200129533
Functional study of TCR-dependent T cell activation in defective T lymphocytes of PNH patients
AUTHOR: Alfinito Fiorella (Reprint); Ruggiero Giuseppina; Andretta Claudia (Reprint); Terrazzano Giuseppe; Zappacosta Serafino; Rotoli Bruno (Reprint)
AUTHOR ADDRESS: Hematology, University of Naples Federico II, Naples, Italy **Italy
JOURNAL: Blood 98 (11 Part 1): p25a November 16, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001; 20011207
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Paroxysmal Nocturnal Hemoglobinuria (PNH) is an acquired clonal disorder of hematopoiesis characterized by hemolytic anemia, thrombophilia and cytopenia. Mutation in the PIG-A gene, responsible for glycosyl-phosphatidyl-inositol (GPI) anchor synthesis, is the hallmark of PNH cells. As a consequence, PNH cells are defective in all GPI membrane-linked proteins. The presence of a residual normal hematopoiesis accounts for the mixed GPI- and GPI+ phenotype, usually found in peripheral blood cells of PNH patients. Functional defects in PNH cells have been already described. In this context, a number of deficiencies in the TCR-dependent signal machinery of GPI- T cells were referred. Physiological TCR-dependent T cell proliferation of naive T lymphocytes needs both MHC-restricted peptide presentation and unrestricted co-stimulatory signals. Moreover the analysis of GPI- T cell responsiveness to defined co-stimulatory pathways is still lacking. In this study we are investigating TCR-dependent T cell proliferation of GPI- lymphocytes in the presence of distinct co-stimulatory signals, as represented by murine transfectants bearing human CD40 or CD80 co-stimulatory molecules. Therefore, fresh GPI- and GPI+ T lymphocytes and short term T cell lines obtained from PNH patients were tested for their ability to proliferate in response to CD3 triggering in the presence of ***CD40*** and ***CD80*** -dependent co-stimulatory pathways. In this model a selective defect in CD40-dependent T cell co-stimulation of GPI- lymphocytes was identified, while the CD80 -dependent pathway appeared to be unaffected. Moreover, no defect in the expression of ***CD40*** ligand (CD154) was ***detected*** . These data

suggest the involvement of GPI-linked proteins in the biological mechanisms underlying distinct TCR-dependent co-stimulatory pathways. Therefore, a role of these molecules in the regulation of naive T lymphocytes proliferation, generally believed to be critically dependent from co-stimulatory signals, might be hypothesized.

10/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16456977 BIOSIS NO.: 200200050488
Detection of mutation in a CD40 ligand gene
AUTHOR: Spriggs M K; Armitage R J; Fanslow W C III
AUTHOR ADDRESS: Seattle, Wash., USA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1191 (3): p1909 Oct. 15, 1996 ***1996***
MEDIUM: print
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Citation
LANGUAGE: English

10/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16413107 BIOSIS NO.: 200200006618
Predictive value of the detection of CD40 and Cytotoxic T-Lymphocytes in early rejecting renal allografts
AUTHOR: Mengel M (Reprint); Mueller I (Reprint); Behrend M; von Woellwarth J; Kreipe H (Reprint)
AUTHOR ADDRESS: Institut fuer Pathologie der Medizinischen Hochschule, Hannover, Germany**Germany
JOURNAL: Kidney and Blood Pressure Research 24 (4-6): p272-273 2001 2001
MEDIUM: print
CONFERENCE/MEETING: Joint Scientific Meeting of the Nephrology Society and the German Working Group for Clinical Nephrology Munster, Germany September 29-October 02, 2001; 20010929
ISSN: 1420-4096
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

10/7/30 (Item 30 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16110760 BIOSIS NO.: 200100282599
Expression of CD23/CD21 and CD40/CD40 ligand in vernal keratoconjunctivitis
AUTHOR: Abu El-Asrar Ahmed M (Reprint); Fatani Rashed A; Missotten Luc; Geboes Karel
AUTHOR ADDRESS: Department of Ophthalmology, King Abdulaziz University Hospital, Airport Road, Riyadh, 11411, Saudi Arabia**Saudi Arabia
JOURNAL: Eye (London) 15 (2): p217-224 April, 2001 2001
MEDIUM: print
ISSN: 0950-222X
DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Purpose: The overproduction of immunoglobulin E (IgE) antibodies is associated with vernal keratoconjunctivitis (VKC). CD23/CD21 and CD40/CD40 ligand (CD40L) interactions have been proposed to be involved in the regulation of IgE synthesis. The purpose of the present study was to investigate the presence and distribution of CD23, CD21, CD40 and CD40L in the conjunctiva from patients with active VKC. Methods: Conjunctival biopsy specimens from 8 subjects with active VKC and 6 control subjects were studied. We used immunohistochemical techniques and a panel of monoclonal antibodies (mAbs) directed against CD23, CD21, CD40 and CD40L. In addition, a panel of mAbs were used to characterise the composition of the inflammatory infiltrate. Results: In the normal conjunctiva, basal epithelial cells and vascular endothelial cells in the upper substantia propria showed a constitutive very weak immunoreactivity for CD40. The immunoreactivity for CD23, CD21 and CD40L was absent. In VKC specimens, the stromal inflammatory infiltrate was orgnanised as a diffuse infiltrate and as small lymphoid follicles consisting of CD20+ B lymphocytes intermingled with smaller numbers of CD3+ T lymphocytes, and CD68+ monocytes/macrophages. Lymphocytes in the centre of the lymphoid follicles showed CD23 and CD21 immunoreactivity. CD40 immunoreactivity in epithelial cells and vascular endothelial cells was stronger in VKC specimens than in control eyes. Furthermore, the majority of mononuclear cells, including T and B lymphocytes, showed immunoreactivity for ***CD40*** . ***CD40L*** immunoreactivity was not ***detected*** . Conclusion: B lymphocytes in the lymphoid follicles expressing CD23, CD21 and CD40 are activated and might be precursors of IgE-producing B cells. These results suggest that the conjunctiva might contribute to IgE synthesis.
? ds

Set	Items	Description
S1	14	E1-E3
S2	4	AU='ROTHE MARTIN'
S3	107	AU='THIEL ANDREAS'
S4	6	(S1 OR S2 OR S3) AND (CD40 OR CD40L RO CD154 OR CD40(W)LIG-AND)
S5	3	RD S4 (unique items)
S6	0	(ANTI(W)CD40 OR CD40)(20N)(DETECT?)(CD4? OR CD8?)
S7	15412	(ANTI(W)CD40 OR CD40)(20N)(DETECT? OR ISOLAT? OR DETERMIN? OR EXPRESS?)(20N)(CD4? OR CD8?)
S8	1098	(ANTI(W)CD40 OR CD40)(10N)(DETECT?)(10N)(CD4? OR CD8?)
S9	567	S8 AND PY<2002
S10	244	RD S9 (unique items)

? t s10/3/51-100

10/3/51 (Item 51 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15701734 BIOSIS NO.: 200000420047
Blockade of T lymphocyte costimulation with cytotoxic T lymphocyte-associated antigen 4-immunoglobulin (CTLA4Ig) reverses the cellular pathology of psoriatic plaques, including the activation of keratinocytes, dendritic cells, and endothelial cells
AUTHOR: Abrams Judith R (Reprint); Kelley Susan L; Hayes Elizabeth; Kikuchi Toyoko; Brown Michael J; Kang Sewon; Lebwohl Mark G; Guzzo Cynthia A; Jegasothy Brian V; Linsley Peter S; Krueger James G
AUTHOR ADDRESS: Novartis Pharmaceuticals Corporation, 59 Route 10, Bldg. 122, Rm. S320, East Hanover, NJ, 07936-1080, USA**USA

JOURNAL: Journal of Experimental Medicine 192 (5): p681-693 September 4, 2000 2000
MEDIUM: print
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/52 (Item 52 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15688663 BIOSIS NO.: 200000406976
Mantle cell lymphoma proliferates upon IL-10 in the CD40 system
AUTHOR: Visser H P J (Reprint); Tewis M; Willemze R; Kluin-Nelemans J C
AUTHOR ADDRESS: Department of Hematology, Laboratory of Experimental Hematology, Leiden University Medical Center, Albinusdreef 2, Building 1, C2-R, 2300 RC, Leiden, Netherlands**Netherlands
JOURNAL: Leukemia (Basingstoke) 14 (8): p1483-1489 August, 2000 2000
MEDIUM: print
ISSN: 0887-6924
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/53 (Item 53 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15651895 BIOSIS NO.: 200000370208
Human decidua contains potent immunostimulatory CD83+ dendritic cells
AUTHOR: Kaemmerer Ulrike (Reprint); Schoppet Michael; McLellan Alexander D; Kapp Michaela; Huppertz Hans-Iko; Kaempgen Eckhart; Dietl Johannes
AUTHOR ADDRESS: Department of Obstetrics and Gynaecology, University of Wuerzburg, Josef-Schneider-Strasse 4, D-97080, Wuerzburg, Germany** Germany
JOURNAL: American Journal of Pathology 157 (1): p159-169 July, 2000 2000
MEDIUM: print
ISSN: 0002-9440
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/54 (Item 54 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15639751 BIOSIS NO.: 200000358064
Up-regulation of BOB.1/OBF.1 expression in normal germinal center B cells and germinal center-derived lymphomas
AUTHOR: Greiner Axel; Mueller Kerstin B; Hess Jochen; Pfeffer Klaus; Mueller-Hermelink H Konrad; Wirth Thomas (Reprint)
AUTHOR ADDRESS: Institut fuer Medizinische Strahlenkunde und Zellforschung, Versbacher Strasse 5, 97078, Wuerzburg, Germany**Germany
JOURNAL: American Journal of Pathology 156 (2): p501-507 February, 2000 2000
MEDIUM: print

ISSN: 0002-9440
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/55 (Item 55 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15618005 BIOSIS NO.: 200000336318
Inhibition of cell growth and Epstein-Barr virus reactivation by CD40 stimulation in Epstein-Barr virus-transformed B cells
AUTHOR: Fukuda Makoto; Satoh Tomohis A; Takanashi Masakatu; Hirai Kanji; Ohnishi Eiko; Sairenji Takeshi (Reprint)
AUTHOR ADDRESS: Department of Biosignaling, School of Life Science, Faculty of Medicine, Tottori University, 86 Nishi-Machi, Yonago, 683-8503, Japan **Japan
JOURNAL: Viral Immunology 13 (2): p215-229 2000 2000
MEDIUM: print
ISSN: 0882-8245
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/56 (Item 56 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15609274 BIOSIS NO.: 200000327587
An increased number of CD40-high monocytes in patients with Crohn's disease
AUTHOR: Sawada-Hase Naoko; Kiyohara Tatsuya (Reprint); Miyagawa Jun-ichiro; Ueyama Harumi; Nishibayashi Hiroyuki; Murayama Yoko; Kashihara Takeshi; Nakahara Masanori; Miyazaki Yoshiji; Kanayama Shuji; Nezu Riichiro; Shinomura Yasuhisa; Matsuzawa Yuji
AUTHOR ADDRESS: Department of Internal Medicine and Molecular Science, Graduate School of Medicine, Osaka University, 2-2 B-5 Yamadaoka, Suita, Osaka, 565-0871, Japan**Japan
JOURNAL: American Journal of Gastroenterology 95 (6): p1516-1523 June, 2000 2000
MEDIUM: print
ISSN: 0002-9270
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/57 (Item 57 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15579936 BIOSIS NO.: 200000298249
Disseminated cytomegalovirus infection as initial manifestation of hyper-IgM syndrome in a 15-month-old boy
AUTHOR: Benesch Martin (Reprint); Pfleger Andreas; Eber Ernst; Orth Ulrike; Zach Maximilian S
AUTHOR ADDRESS: Klinische Abteilung fuer Paediatrische Pulmonologie und Allergologie, Universitaetsklinik fuer Kinder-und Jugendheilkunde, Auenbruggerplatz 30, 8036, Graz, Austria**Austria
JOURNAL: European Journal of Pediatrics 159 (6): p453-455 June, 2000

2000
MEDIUM: print
ISSN: 0340-6199
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/58 (Item 58 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15578653 BIOSIS NO.: 200000296966
Early myeloid cells are high producers of nitric oxide upon CD40 plus IFN-gamma stimulation through a mechanism dependent on endogenous TNF-alpha and IL-1alpha
AUTHOR: Angulo Inigo; Rullas Joaquin; Campillo Jose Antonio; Obregon Eva; Heath Andrew; Howard Maureen; Munoz-Fernandez Maria Angeles; Subiza Jose Luis (Reprint)
AUTHOR ADDRESS: Department of Immunology, Hospital Clinico San Carlos, E-28040, Madrid, Spain**Spain
JOURNAL: European Journal of Immunology 30 (5): p1263-1271 May, 2000
2000
MEDIUM: print
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/59 (Item 59 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15509918 BIOSIS NO.: 200000228231
Effect of interleukin-7 on the in vitro development and maturation of monocyte derived human dendritic cells
AUTHOR: Li L Q; Masucci M G; Levitsky V (Reprint)
AUTHOR ADDRESS: Microbiology and Tumour Biology Center, Karolinska Institutet, S-171 77, Stockholm, Sweden**Sweden
JOURNAL: Scandinavian Journal of Immunology 51 (4): p361-371 April, 2000
2000
MEDIUM: print
ISSN: 0300-9475
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/60 (Item 60 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15485538 BIOSIS NO.: 200000203851
High sequence homology between human and pig CD40 with conserved binding to human CD154
AUTHOR: Rushworth Stuart A; Bravery Christopher A; Thompson Simon (Reprint)
AUTHOR ADDRESS: Molecular Biology Unit, Imutran Limited (A Novartis Pharma AG Company), Cambridge, CB2 2YP, UK**UK
JOURNAL: Transplantation (Baltimore) 69 (5): p936-940 March 15, 2000
2000

MEDIUM: print
ISSN: 0041-1337
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/61 (Item 61 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15473528 BIOSIS NO.: 200000191841
The formation of immunogenic major histocompatibility complex class II-peptide ligands in lysosomal compartments of dendritic cells is regulated by inflammatory stimuli
AUTHOR: Inaba Kayo; Turley Shannon; Iyoda Tomonori; Yamaide Fumiya; Shimoyama Susumu; Reis e Sousa Caetano; Germain Ronald N; Mellman Ira; Steinman Ralph M (Reprint)
AUTHOR ADDRESS: Laboratory of Cell Physiology and Immunology, The Rockefeller University, 1230 York Ave., 405 Bronk Bldg., New York, NY, 10021-6399, USA**USA
JOURNAL: Journal of Experimental Medicine 191 (6): p927-936 March 20, 2000 2000
MEDIUM: print
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/62 (Item 62 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15446577 BIOSIS NO.: 200000164890
Immunostimulatory CpG-oligonucleotides cause proliferation, cytokine production, and an immunogenic phenotype in chronic lymphocytic leukemia B cells
AUTHOR: Decker Thomas; Schneller Folker; Sparwasser Tim; Tretter Theresa; Lipford Grayson B; Wagner Hermann; Peschel Christian (Reprint)
AUTHOR ADDRESS: IIIrd Department of Medicine, Technical University of Munich, Ismaninger Str. 15, 81675, Munich, Germany**Germany
JOURNAL: Blood 95 (3): p999-1006 Feb. 1, 2000 ***2000***
MEDIUM: print
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/63 (Item 63 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15438815 BIOSIS NO.: 200000157128
CD4+ T cells play an important role in acute experimental pancreatitis in mice
AUTHOR: Demols Anne; Le Moine Olivier; Desalle Fabrice; Quertinmont Eric; Van Laethem Jean-Luc; Deviere Jacques (Reprint)
AUTHOR ADDRESS: Department of Gastroenterology, Hopital Erasme, ULB, Route de Lennik, 808, B 1070, Brussels, Belgium**Belgium

JOURNAL: Gastroenterology 10 (5): p582-590 March 9, 2000 2000
MEDIUM: print
ISSN: 0016-5085
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/64 (Item 64 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15396207 BIOSIS NO.: 200000114520
Immunomodulatory dendritic cells generated from nonfractionated bulk peripheral blood mononuclear cell cultures induce growth of cytotoxic T cells against renal cell carcinoma
AUTHOR: Hinkel Andreas; Tso Cho-Lea; Gitlitz Barbara J; Neagos Negoita; Schmid Ingrid; Paik Sun H; deKernion Jean; Figlin Robert; Belldegrun Arie (Reprint)
AUTHOR ADDRESS: Department of Urology, UCLA School of Medicine, CHS 66-118, Los Angeles, CA, 90095-1738, USA**USA
JOURNAL: Journal of Immunotherapy 23 (1): p83-93 Jan., 2000 ***2000***
MEDIUM: print
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/65 (Item 65 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15396174 BIOSIS NO.: 200000114487
Cell-free recombination of immunoglobulin switch-region DNA with nuclear extracts
AUTHOR: Zhang Ke (Reprint); Cheah Hai-Kit
AUTHOR ADDRESS: Division of Clinical Immunology/Allergy, Department of Medicine, UCLA School of Medicine, 52-175, CHS, Los Angeles, CA, 90095-1680, USA**USA
JOURNAL: Clinical Immunology (Orlando) 94 (2): p140-151 Feb., 2000 2000
MEDIUM: print
ISSN: 1521-6616
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/66 (Item 66 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15390364 BIOSIS NO.: 200000108677
Pararosaniline fixation for detection of co-stimulatory molecules, cytokines, and specific antibody
AUTHOR: Schrijver Ingrid A (Reprint); Melief Marie-Jose; van Meurs Marjan; Companjen Arjen R; Laman Jon D
AUTHOR ADDRESS: Dept. of Immunology, Erasmus University Rotterdam, 3000 DR, Rotterdam, Netherlands**Netherlands
JOURNAL: Journal of Histochemistry and Cytochemistry 48 (1): p95-103 Jan., 2000 2000

MEDIUM: print
ISSN: 0022-1554
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/67 (Item 67 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15375927 BIOSIS NO.: 200000094240
Development of dendritic cells in vitro from murine fetal liver-derived lineage phenotype-negative c-kit+ hematopoietic progenitor cells
AUTHOR: Zhang Yanyun; Zhang Yi; Wang Yong; Ogata Masafumi; Hashimoto Shin-ichi; Onai Nobuyuki; Matsushima Kouji (Reprint)
AUTHOR ADDRESS: Department of Molecular Preventive Medicine, School of Medicine, University of Tokyo, 7-3-1, Hongo, Bunkyo-ku, Tokyo, 113-0033, Japan**Japan
JOURNAL: Blood 95 (1): p138-146 Jan. 1, 2000 ***2000***
MEDIUM: print
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/68 (Item 68 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15321415 BIOSIS NO.: 200000039728
Expression of CD40 and its ligand, CD40L, in intestinal lesions of Crohn's disease
AUTHOR: Battaglia Edda; Biancone Luigi; Resegotti Andrea; Emanuelli Giorgio ; Fronda Gian Ruggero; Camussi Giovanni (Reprint)
AUTHOR ADDRESS: Cattedra di Nefrologia, Dipartimento di Medicina Interna, Universita di Torino, Corso Dogliotti 14, 10126, Torino, Italy**Italy
JOURNAL: American Journal of Gastroenterology 94 (11): p3279-3284 Nov., 1999 1999
MEDIUM: print
ISSN: 0002-9270
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/69 (Item 69 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15309937 BIOSIS NO.: 200000028250
Myasthenia gravis: Selective enrichment of anti-acetylcholine receptor antibody production in untransformed human B cell cultures
AUTHOR: Padberg Frank; Matsuda Masayuki; Fenk Roland; Patenge Nadja; Kubuschok Boris; Hohlfeld Reinhard; Wekerle Hartmut; Spuler Simone (Reprint)
AUTHOR ADDRESS: Department of Psychiatry, University of Munich, Nussbaumstrasse 7, D-80336, Munich, Germany**Germany
JOURNAL: European Journal of Immunology 29 (11): p3538-3548 Nov., 1999 1999

MEDIUM: print
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/70 (Item 70 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15309935 BIOSIS NO.: 200000028248
CD40 associates with the MHC class II molecules on human B cells
AUTHOR: Leveille Claire; Chandad Fatiha; Al-Daccak Reem; Mourad Walid (Reprint)
AUTHOR ADDRESS: Centre de Recherche en Rhumatologie et Immunologie, Centre Hospitalier de l''Universite Laval, 2705 Boulevard Laurier, Sainte-Foy, PQ, G1V 4G2, Canada**Canada
JOURNAL: European Journal of Immunology 29 (11): p3516-3526 Nov., 1999 1999
MEDIUM: print
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/71 (Item 71 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15284852 BIOSIS NO.: 200000003165
Human hematopoietic stem/progenitor cells generate CD5+ B lymphoid cells in NOD/SCID mice
AUTHOR: Novelli Enrico M; Ramirez Manuel; Leung Wing; Civin Curt I (Reprint)
AUTHOR ADDRESS: Oncology 3-109, Johns Hopkins Hospital, 600 N. Wolfe Street, Baltimore, MD, 21287-5001, USA**USA
JOURNAL: Stem Cells (Miamisburg) 17 (5): p242-252 1999 1999
MEDIUM: print
ISSN: 1066-5099
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/72 (Item 72 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15281298 BIOSIS NO.: 199900540958
Expression of functional CD40 in human hepatocellular carcinoma
AUTHOR: Sugimoto Kazushi; Shiraki Katsuya (Reprint); Ito Takeshi; Fujikawa Katsuhiko; Takase Koujiro; Tameda Yukihiko; Moriyama Masami; Nakano Takeshi
AUTHOR ADDRESS: First Department of Internal Medicine, Mie University School of Medicine, 2-174 Edobashi, Tsu, 514-8507, Japan**Japan
JOURNAL: Hepatology 30 (4): p920-926 Oct., 1999 ***1999***
MEDIUM: print
ISSN: 0270-9139
DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: English

10/3/73 (Item 73 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15238664 BIOSIS NO.: 199900498324
Isolation and characterization of macaque dendritic cells from CD34+ bone marrow progenitors
AUTHOR: Pinchuk Lesya M; Grouard-Vogel Geraldine; Magaletti Dario M; Doty Raymond T; Andrews Robert G; Clark Edward A (Reprint)
AUTHOR ADDRESS: Regional Primate Research Center, University of Washington, Seattle, WA, USA**USA
JOURNAL: Cellular Immunology 196 (1): p34-40 Aug. 25, 1999 ***1999***
MEDIUM: print
ISSN: 0008-8749
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/74 (Item 74 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15235085 BIOSIS NO.: 199900494745
Dietary lipid lowering reduces tissue factor expression in rabbit atheroma
AUTHOR: Aikawa Masanori (Reprint); Voglic Sami J; Sugiyama Seigo; Rabkin Elena; Taubman Mark B; Fallon John T; Libby Peter
AUTHOR ADDRESS: Vascular Medicine and Atherosclerosis Unit, Brigham and Women's Hospital, Harvard Medical School, 221 Longwood Ave, LMRC 309, Boston, MA, 02115, USA**USA
JOURNAL: Circulation 100 (11): p1215-1222 Sept. 14, 1999 ***1999***
MEDIUM: print
ISSN: 0009-7322
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/75 (Item 75 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15220707 BIOSIS NO.: 199900480367
FADD expression and caspase activation in B-cell lymphomas resistant to Fas-mediated apoptosis
AUTHOR: Xerri Luc (Reprint); Devilard Elisabeth; Bouabdallah Reda; Stoppa Anne-Marie; Hassoun Jacques; Birg Francoise
AUTHOR ADDRESS: Departement de Pathologie, INSERM U119, Institut Paoli-Calmettes, 232 Boulevard de Sainte Marguerite, 13273, Marseille Cedex 9, France**France
JOURNAL: British Journal of Haematology 106 (3): p652-661 Sept., 1999
1999
MEDIUM: print
ISSN: 0007-1048
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/76 (Item 76 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15136459 BIOSIS NO.: 199900396119
Differential requirements for tumor necrosis factor receptor-associated factor family proteins in CD40-mediated induction of NF-kappaB and Jun N-terminal kinase activation
AUTHOR: Leo Eugen; Welsh Kate; Matsuzawa Shu-ichi; Zapata Juan M; Kitada Shinichi; Mitchell Richard S; Ely Kathryn R; Reed John C (Reprint)
AUTHOR ADDRESS: Burnham Institute, 10901 N. Torrey Pines Rd., La Jolla, CA, 92037, USA**USA
JOURNAL: Journal of Biological Chemistry 274 (32): p22414-22422 Aug. 6, 1999 1999
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/77 (Item 77 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15101174 BIOSIS NO.: 199900360834
Reactive plasmacytoses are expansions of plasmablasts retaining the capacity to differentiate into plasma cells
AUTHOR: Jego Gaetan; Robillard Nelly; Puthier Denis; Amiot Martine; Accard Francoise; Pineau Danielle; Harousseau Jean-Luc; Bataille Regis; Pellat-Deceunynck Catherine (Reprint)
AUTHOR ADDRESS: INSERM U463, Institut de Biologie, 9, quai Moncousu, 44093, Nantes Cedex 01, France**France
JOURNAL: Blood 94 (2): p701-712 July, 1999 1999
MEDIUM: print
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/78 (Item 78 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15097964 BIOSIS NO.: 199900357624
Immune responses to Plasmodium falciparum-merozoite surface protein 1 (MSP1) antigen, II. Induction of parasite-specific immunoglobulin G in unsensitized human B cells after in vitro T-cell priming with MSP119
AUTHOR: Garraud O (Reprint); Diouf A; Holm I; Perraut R; Longacre S
AUTHOR ADDRESS: Unite d'Immunologie, Institut Pasteur, 36 Avenue Pasteur, Dakar, Senegal**Senegal
JOURNAL: Immunology 97 (3): p497-505 July, 1999 1999
MEDIUM: print
ISSN: 0019-2805
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/79 (Item 79 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15087878 BIOSIS NO.: 199900347538
Soluble CD40 in the serum of healthy donors, patients with chronic renal failure, haemodialysis and chronic ambulatory peritoneal dialysis (CAPD) patients
AUTHOR: Schwabe R F; Engelmann H; Hess S; Fricke H (Reprint)
AUTHOR ADDRESS: Department of Medicine, University of Munich, Ziemssenstrasse 1, 80336, Munich, Germany**Germany
JOURNAL: Clinical and Experimental Immunology 117 (1): p153-158 July, 1999 1999
MEDIUM: print
ISSN: 0009-9104
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/80 (Item 80 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15073843 BIOSIS NO.: 199900333503
An aggressive form of polyarticular arthritis in a man with CD154 mutation (X-linked hyper-IgM syndrome)
AUTHOR: Webster Elizabeth A; Khakoo Aarif Y; Mackus Wendeline JM; Karpusas Michael; Thomas David W; Davidson Anne; Christian Charles L; Lederman Seth (Reprint)
AUTHOR ADDRESS: Laboratory of Molecular Immunology, Columbia University, 630 West 168th Street, PH8-405, New York, NY, 10032, USA**USA
JOURNAL: Arthritis and Rheumatism 42 (6): p1291-1296 June, 1999 1999
MEDIUM: print
ISSN: 0004-3591
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/81 (Item 81 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15052429 BIOSIS NO.: 199900312089
Characterization of CD40-dependent immunoglobulin class switching
AUTHOR: Strom L (Reprint); Laurencikiene J; Miskiniene A; Severinson E
AUTHOR ADDRESS: Department of Cell and Molecular Biology, Karolinska Institute, S-171 77, Stockholm, Sweden**Sweden
JOURNAL: Scandinavian Journal of Immunology 49 (5): p523-532 May, 1999 1999
MEDIUM: print
ISSN: 0300-9475
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/82 (Item 82 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15043360 BIOSIS NO.: 199900303020
Human B cells accumulate immunoglobulin V gene somatic mutations in a cell contact-dependent manner in cultures supported by activated T cells but not in cultures supported by CD40 ligand
AUTHOR: Huang S-C; Glas A M; Pinchuk G V; Van Montfort E H N; Rao S P; Jiang R; Milner E C B (Reprint)
AUTHOR ADDRESS: Immunology Program, Virginia Mason Research Center, 1000 Seneca Street, Seattle, WA, 98101, USA**USA
JOURNAL: Clinical and Experimental Immunology 116 (3): p441-448 June, 1999 1999
MEDIUM: print
ISSN: 0009-9104
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/83 (Item 83 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15041127 BIOSIS NO.: 199900300787
Production of IL-4 and expression of CD40 ligand by human CD8+T cells
AUTHOR: Yanagihara Yukiyoshi (Reprint); Kajiwara Keiichi; Koshio Takehiro; Basaki Yuji; Ikizawa Koichi; Mori Miyuki; Akiyama Kazuo; Kawamura Nobuaki ; Sakiyama Yukio
AUTHOR ADDRESS: Clinical Research Center for Allergy, National Sagamihara Hospital, 18-1 Sakuradai, Sagamihara City, Kanagawa, 228-8522, Japan** Japan
JOURNAL: Journal of Allergy and Clinical Immunology 103 (5 PART 2): p S405-S411 May, 1999 1999
MEDIUM: print
ISSN: 0091-6749
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/84 (Item 84 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15039255 BIOSIS NO.: 199900298915
Enhanced expression of CD80 (B7-1), CD86 (B7-2), and CD40 and their ligands CD28 and CD154 in fulminant hepatic failure
AUTHOR: Leifeld Ludger (Reprint); Trautwein Christian; Dumoulin Franz Ludwig; Manns Michael Peter; Sauerbruch Tilman; Spengler Ulrich
AUTHOR ADDRESS: Department of Internal Medicine, University of Bonn, Sigmund Freud Strasse 25, D-53105, Bonn, Germany**Germany
JOURNAL: American Journal of Pathology 154 (6): p1711-1720 June, 1999 1999
MEDIUM: print
ISSN: 0002-9440
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/85 (Item 85 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

15012691 BIOSIS NO.: 199900272351
In situ expression of B7 and CD40 costimulatory molecules by normal human lung macrophages and epithelioid cells in tuberculoid granulomas
AUTHOR: Soler P (Reprint); Boussaud V; Moreau J; Bergeron A; Bonnette P; Hance A J; Tazi A
AUTHOR ADDRESS: Faculte Xavier Bichat, INSERM U82, 75870, Paris cedex 18, France**France
JOURNAL: Clinical and Experimental Immunology 116 (2): p332-339 May, 1999 1999
MEDIUM: print
ISSN: 0009-9104
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/86 (Item 86 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14978068 BIOSIS NO.: 199900237728
CD40 expression by human bronchial epithelial cells
AUTHOR: Gormand F (Reprint); Briere F; Peyrol S; Raccurt M; Durand I; Ait-Yahia S; Lebecque S; Banchereau J; Pacheco Y
AUTHOR ADDRESS: Centre Hospitalier Lyon-Sud, Pavillon 5F, 69310, Pierre Benite, France**France
JOURNAL: Scandinavian Journal of Immunology 49 (4): p355-361 April, 1999 1999
MEDIUM: print
ISSN: 0300-9475
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/87 (Item 87 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14973518 BIOSIS NO.: 199900233178
Inhibition of human breast carcinoma growth by a soluble recombinant human CD40 ligand
AUTHOR: Hirano Akio; Longo Dan L; Taub Dennis D; Ferris Douglas K; Young Lawrence S; Eliopoulos Arisitides G; Agathanggelou Angelo; Cullen Nicky; Macartney James; Fanslow William C; Murphy William J (Reprint)
AUTHOR ADDRESS: SAIC-Frederick, Bldg 567, Room 210, Frederick, MD, USA**USA
JOURNAL: Blood 93 (9): p2999-3007 May 1, 1999 1999
MEDIUM: print
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/88 (Item 88 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14953084 BIOSIS NO.: 199900212744
HepG2 human hepatoma cells express multiple cytokine genes
AUTHOR: Stonans Ilmars; Stonane Elita; Russwurm Stefan (Reprint); Deigner Hans-Peter; Boehm Konrad J; Wiederhold Matthias; Jaeger Lothar; Reinhart Konrad
AUTHOR ADDRESS: Clinic of Anesthesiology and Intensive Therapy, Friedrich-Schiller-University of Jena, D-07740, Jena, Germany**Germany
JOURNAL: Cytokine 11 (2): p151-156 Feb., 1999 ***1999***
MEDIUM: print
ISSN: 1043-4666
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/89 (Item 89 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14947223 BIOSIS NO.: 199900206883
CD40 expression on graft infiltrates and parenchymal CD154 (CD40L) induction in human chronic renal allograft rejection
AUTHOR: Gaweco Anderson S (Reprint); Mitchell Bonnie L; Lucas Bruce A; McClatchey Kenneth D; Van Thiel David H
AUTHOR ADDRESS: Liver Transplant Program, Loyola University Medical Center, Loyola University Chicago, 2160 South First Avenue, Maywood, IL, 60153, USA**USA
JOURNAL: Kidney International 55 (4): p1543-1552 April, 1999 1999
MEDIUM: print
ISSN: 0085-2538
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/90 (Item 90 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14930011 BIOSIS NO.: 199900189671
Isolation of MUC1-primed B lymphocytes from tumour-draining lymph nodes by immunomagnetic beads
AUTHOR: Petrarca Claudia; Casalino Beniamino; von Mensdorff-Pouilly Silvia; Rughetti Aurelia; Rahimi Hassan; Scambia Giovanni; Hilgers Joseph; Frati Luigi; Nuti Marianna (Reprint)
AUTHOR ADDRESS: Department of Experimental Medicine and Pathology, University of Rome, Viale Regina Elena 324, I-00161, Rome, Italy**Italy
JOURNAL: Cancer Immunology Immunotherapy 47 (5): p272-277 Jan., 1999 1999
MEDIUM: print
ISSN: 0340-7004
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/91 (Item 91 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14884242 BIOSIS NO.: 199900143902

Control of apoptosis in Epstein Barr virus-positive nasopharyngeal carcinoma cells: Opposite effects of CD95 and CD40 stimulation
AUTHOR: Sbih-Lammali Fatima; Clausse Bernard; Ardila-Osorio Hector; Guerry Roland; Talbot Monique; Havouis Severine; Ferradini Laurent; Bosq Jacques ; Fursz Thomas; Busson Pierre (Reprint)
AUTHOR ADDRESS: Lab. Biol. Tumeurs Humaines, Inst. Gustave Roussy, 94805 Villejuif Cedex, France**France
JOURNAL: Cancer Research 59 (4): p924-930 Feb. 15, 1999 ***1999***
MEDIUM: print
ISSN: 0008-5472
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/92 (Item 92 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14852510 BIOSIS NO.: 199900112170
CD40 activation induces apoptosis in cultured human hepatocytes via induction of cell surface Fas ligand expression and amplifies Fas-mediated hepatocytes death during allograft rejection
AUTHOR: Afford Simon C (Reprint); Randhawa Satinder; Eliopoulos Aristides G ; Hubscher Stefan G; Young Lawrence S; Adams David H
AUTHOR ADDRESS: Liver Res. Labs., Queen Elizabeth Hosp., Edgbaston, Birmingham B15 2TH, UK**UK
JOURNAL: Journal of Experimental Medicine 189 (2): p441-446 Jan. 18, 1999 1999
MEDIUM: print
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/93 (Item 93 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14798617 BIOSIS NO.: 199900058277
Human B cell differentiation: Dependence on interactions with monocytes and T lymphocytes via CD40, CD80 (B7.1), and the CD2-ligands CD48 and CD58 (LFA-3)
AUTHOR: Hoffmann Joerg C (Reprint); Krueger Hartmut; Zielen Stefan; Bayer Bettina; Zeidler Henning
AUTHOR ADDRESS: Abt. Rheumatol., Zentrum Innere Med. und Dermatol., Med. Hochschule Hannover, 30623 Hannover, Germany**Germany
JOURNAL: Cell Biology International 22 (1): p21-29 1998 1998
MEDIUM: print
ISSN: 1065-6995
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/94 (Item 94 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14747043 BIOSIS NO.: 199900006703

Generation of functional human dendritic cells from adherent peripheral blood monocytes by CD40 ligation in the absence of granulocyte-macrophage colony-stimulating factor
AUTHOR: Brossart Peter; Gruenebach Frank; Stuhler Gernot; Reichardt Volker L; Moehle Robert; Kanz Lothar; Brugger Wolfram (Reprint)
AUTHOR ADDRESS: Dep. Hematol. Oncol. Immunol., Univ. Tuebingen, Otfried-Mueller-Strasse 10, D-72076 Tuebingen, Germany**Germany
JOURNAL: Blood 92 (11): p4238-4247 Dec. 1, 1998 ***1998***
MEDIUM: print
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/95 (Item 95 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14718777 BIOSIS NO.: 199800513024
Coexpression of CD40 and CD40 ligand in B-cell lymphoma cells
AUTHOR: Clodi Katharina; Asgary Zahra; Zhao Shourong; Kliche Kay-Oliver; Cabanillas Fernando; Andreeff Michael; Younes Anas (Reprint)
AUTHOR ADDRESS: Dep. Lymphoma, Univ. Texas M.D. Anderson Cancer Cent., 1515 Holcombe Blvd., Houston, TX 77030, USA**USA
JOURNAL: British Journal of Haematology 103 (1): p270-275 Oct., 1998 1998
MEDIUM: print
ISSN: 0007-1048
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/96 (Item 96 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14704436 BIOSIS NO.: 199800498683
CD40 is functionally expressed on human breast carcinomas: Variable inducibility by cytokines and enhancement of Fas-mediated apoptosis
AUTHOR: Wingett Denise G; Vestal Robert E; Forcier Kristin; Hadjokas Nicholas; Nielson Christopher P (Reprint)
AUTHOR ADDRESS: Res. Serv. (151), VA Med. Cent., 500 W. Fort Street, Boise, ID 83702, USA**USA
JOURNAL: Breast Cancer Research and Treatment 50 (1): p27-36 July, 1998 1998
MEDIUM: print
ISSN: 0167-6806
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/97 (Item 97 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14703829 BIOSIS NO.: 199800498076
Prolonged phenotypic, functional, and molecular change in group I Burkitt lymphoma cells on short-term exposure to CD40 ligand

AUTHOR: Baker Matthew P; Eliopoulos Aristides G; Young Lawrence S; Armitage Richard J; Gregory Christopher D; Gordon John (Reprint)
AUTHOR ADDRESS: Dep. Immunol. Med. Sch., Univ. Birmingham, Vincent Drive, Edgbaston, Birmingham B15 2TT, UK**UK
JOURNAL: Blood 92 (8): p2830-2843 Oct. 15, 1998 ***1998***
MEDIUM: print
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/98 (Item 98 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14642989 BIOSIS NO.: 199800437236
Therapy with antibodies against CD40L (CDI54) and CD44-variant isoforms reduces experimental autoimmune encephalomyelitis induced by a proeteolipid protein peptide
AUTHOR: Laman J D (Reprint); Maassen C B M; Schellekens M M; Visser L; Kap M; De Jong E; Van Puijenbroek M; Van Stipdonk M J B; Van Meurs M; Schwarzler C; Gunthert U
AUTHOR ADDRESS: Div. Immunological Infect. Dis., TNO Prevention Health, PO Box 2215, 2301 CE Leiden, Netherlands**Netherlands
JOURNAL: Multiple Sclerosis 4 (3): p147-153 June, 1998 1998
MEDIUM: print
ISSN: 1352-4585
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/99 (Item 99 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14640130 BIOSIS NO.: 199800434377
B cell-mediated down-regulation of IFN-gamma and IL-12 production induced during anti-tumor immune responses in the tumor-bearing state
AUTHOR: Wijesuriya Rishani; Maruo Seiji; Zou Jian-Ping; Ogawa Makoto; Umehara Kazunari; Yamashita Motozo; Ono Shiro; Fujiwara Hiromi (Reprint); Hamaoka Toshiyuki
AUTHOR ADDRESS: Biomed. Res. Cent., Osaka Univ. Med. Sch., 2-2 Yamada-Oka, Suita, Osaka 565, Japan**Japan
JOURNAL: International Immunology 10 (8): p1057-1065 Aug., 1998 ***1998***
MEDIUM: print
ISSN: 0953-8178
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

10/3/100 (Item 100 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14571524 BIOSIS NO.: 199800365771
Role of the endogenous production of interleukin 12 in immunotherapy
AUTHOR: Harada Mamoru (Reprint); Tamada Koji; Abe Koichiro; Yasumoto Kosei; Kimura Genki; Nomoto Kikuo

AUTHOR ADDRESS: Surgery Branch, Natl. Cancer Inst., NIH, Build. 10, Room 2B42, Bethesdsa, MD 20892, USA**USA
JOURNAL: Cancer Research 58 (14): p3073-3077 July 15, 1998 1998
MEDIUM: print
ISSN: 0008-5472
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
? t s10/7,68,83,89,93
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>>>Format 93 is not valid in file 73
>>>Format 93 is not valid in file 155
>>>Format 93 is not valid in file 399
? t s10/7/68,83,89,93

10/7/68 (Item 68 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15321415 BIOSIS NO.: 200000039728
Expression of CD40 and its ligand, CD40L, in intestinal lesions of Crohn's disease
AUTHOR: Battaglia Edda; Biancone Luigi; Resegotti Andrea; Emanuelli Giorgio ; Fronda Gian Ruggero; Camussi Giovanni (Reprint)
AUTHOR ADDRESS: Cattedra di Nefrologia, Dipartimento di Medicina Interna, Universita di Torino, Corso Dogliotti 14, 10126, Torino, Italy**Italy
JOURNAL: American Journal of Gastroenterology 94 (11): p3279-3284 Nov., 1999 1999
MEDIUM: print
ISSN: 0002-9270
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: OBJECTIVE: Selected mechanisms of the immune system participate in the development of inflammatory bowel disease. Recently, overexpression of the ligand for CD40 (CD40L), a lymphocyte costimulatory molecule, was shown to induce severe inflammatory bowel disease in transgenic mice. In the present study, we examined the expression of CD40 and CD40L on surgical specimens of ileum from 12 patients with Crohn's disease and 10 patients with diverticulitis. METHODS: Several CD40L+ cells were present in the affected tissue of patients with Crohn's disease, whereas few scattered CD40L+ cells were detected in sections of histologically normal ileum, resected distantly from the affected tissue, in patients with diverticulitis and in normal ileum portions obtained from colorectal cancer undergoing extensive surgery. The phenotype of ***CD40L*** + cells was mainly ***CD4*** +. RESULTS: In patients with Crohn's disease, several ***CD40*** + cells were detectable in the same areas of lymphocytes expressing CD40L, whereas in patients with diverticulitis, the number of ***CD40*** + cells was significantly lower. Most of the ***CD40*** + cells costained with CD20, thus showing to be B-lymphocytes, and only a few were CD14+ macrophages. Several von Willebrand-positive vessels were also positive for CD40. In addition, several infiltrating macrophages were found to express B7-1 and B7-2 molecules, the ligands of CD28 and CTLA-4, which cooperate with the CD40-CD40L pathway in lymphocyte activation. Staining of ileal lesions with anti-CTLA-4 antibodies resulted in ***detection*** of none or very few positive cells. In contrast, in patients with diverticulitis, an enhanced number of B7-1 and B7-2 and CTLA-4 was observed. CONCLUSION: The local accumulation of CD40L+ together with CD40+ cells within intestinal lesions of Crohn's

disease suggests the involvement of this co-stimulatory pathway.

10/7/83 (Item 83 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15041127 BIOSIS NO.: 199900300787
Production of IL-4 and expression of CD40 ligand by human CD8+T cells
AUTHOR: Yanagihara Yukiyoshi (Reprint); Kajiwara Keiichi; Koshio Takehiro; Basaki Yuji; Ikizawa Koichi; Mori Miyuki; Akiyama Kazuo; Kawamura Nobuaki ; Sakiyama Yukio
AUTHOR ADDRESS: Clinical Research Center for Allergy, National Sagamihara Hospital, 18-1 Sakuradai, Sagamihara City, Kanagawa, 228-8522, Japan** Japan
JOURNAL: Journal of Allergy and Clinical Immunology 103 (5 PART 2): p S405-S411 May, 1999 1999
MEDIUM: print
ISSN: 0091-6749
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: The role of CD8+ T cells in IgE synthesis remains unclear. Objective: The aim of this study was to investigate IL-4 production and CD40 ligand expression by human CD8+ T cells. Methods: We conducted functional and phenotypic analyses of human T cells in peritoneal washings from severe combined immunodeficiency mice reconstituted with PBMCs from normal and atopic human donors. We also examined the expression of IL-4 and CD40 ligand by CD8+ T cells from a patient with adenosine deaminase deficiency who received autologous T cell-directed gene therapy. Results: Transfer of atopic cells into the mice caused production of IgE and IgG with increased expression of IL-4 and ***CD40*** ligand mRNA. In addition, both intracellular IL-4 and cell surface CD40 ligand were detected in CD8+ and in ***CD4*** + T cells. ***CD8*** + T-cell lines generated from the patient's T cells carrying the adenosine deaminase gene expressed not only IL-4 mRNA and protein but also CD40 ligand mRNA and protein after being stimulated with an anti-CD3 mAb. After anti-CD3 stimulation and paraformaldehyde fixation, CD8+ T cells induced IgE synthesis by normal human B cells in the presence of recombinant IL-4. Conclusion: Taken together, these results demonstrate that IL-4-producing and CD40 ligand-expressing CD8+ cells are detectable among human T cells and suggest that such cells may promote IgE production by B cells under some conditions.

10/7/89 (Item 89 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14947223 BIOSIS NO.: 199900206883
CD40 expression on graft infiltrates and parenchymal CD154 (CD40L) induction in human chronic renal allograft rejection
AUTHOR: Gaweco Anderson S (Reprint); Mitchell Bonnie L; Lucas Bruce A; McClatchey Kenneth D; Van Thiel David H
AUTHOR ADDRESS: Liver Transplant Program, Loyola University Medical Center, Loyola University Chicago, 2160 South First Avenue, Maywood, IL, 60153, USA**USA
JOURNAL: Kidney International 55 (4): p1543-1552 April, 1999 1999
MEDIUM: print

ISSN: 0085-2538
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background. CD40-CD154 (CD40L) costimulatory signaling plays a pivotal role in the effector mechanisms of transplant graft rejection. In animal models, CD40-CD154 blockade induces long-term graft acceptance concurrent with an absence of chronic rejection (CR) lesions. Given the critical importance of CD40-CD154 interactions in the development of chronic transplant allograft rejection, the relevance of in situ CD40 and CD154 expression was assessed in human chronic renal allograft rejection. Methods. The expression of CD40, CD154, CD68, and T-cell receptor (TCR)alpha/beta was analyzed by immunohistochemistry. Serial cryostat sections of snap-frozen core renal allograft biopsies were obtained from 30 renal transplant patients. Biopsy specimens received diagnoses of CR (N = 23) according to the Banff classification and were compared with controls (N = 7) consisting of stable allografts and normal kidney tissue. Results. Striking CD40 staining of graft cellular infiltrates (P = 0.016) was observed in renal allografts with CR compared with controls. The CD40+ cellular infiltrates in CR were predominantly TCRalpha/beta+ T cells and some CD68+ macrophages. These findings were contrasted by the low-level CD40 expression detected in glomeruli and tubules of CR and controls. However, glomerular induction of CD154 was observed in CR allografts (P = 0.028) as compared with controls. CD154 immunoreactivity was demonstrated on glomerular endothelial, epithelial, and mesangial cells. Moderate CD154 expression was detected on tubular epithelial cells, and only weak CD154 immunoreactivity was observed on the infiltrates in isolated CR cases. Conclusion. In human chronic renal allograft rejection, CD40 is expressed on graft-infiltrating cells of the T cell and macrophage compartments. CD154 expression is induced on glomerular and tubular epithelial cells during CR, demonstrating another novel source of CD154 expression. The data substantiate the potential contributory role of an interaction between CD40+ graft-destructive effector T cells and macrophages with CD154+ renal allograft parenchymal cells in the development of chronic renal allograft rejection.

14798617 BIOSIS NO.: 199900058277
Human B cell differentiation: Dependence on interactions with monocytes and T lymphocytes via CD40, CD80 (B7.1), and the CD2-ligands CD48 and CD58 (LFA-3)
AUTHOR: Hoffmann Joerg C (Reprint); Krueger Hartmut; Zielen Stefan; Bayer Bettina; Zeidler Henning
AUTHOR ADDRESS: Abt. Rheumatol., Zentrum Innere Med. und Dermatol., Med. Hochschule Hannover, 30623 Hannover, Germany**Germany
JOURNAL: Cell Biology International 22 (1): p21-29 1998 1998
MEDIUM: print
ISSN: 1065-6995
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: B cell differentiation depends on cellular interactions with T lymphocytes and monocytes via adhesion molecules (AM). In order to characterize AM which are required for B cell differentiation immunoglobulin production using unseparated peripheral blood mononuclear

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  cells (PBMC) was studied. Unstimulated human PBMC were cultured for 9
  days with mAb directed at CD2/CD48, /CD58, CD59, CD5/CD72,
  CD11a-CD18/CD54, CD28/CD80, CD86, CD40/CD40L, or rat CD2 (control). B
  cell differentiation was quantified measuring IgM and in some cases IgA,
  IgG, and IgE production. IgM levels were significantly reduced by mAb
  against   ***CD40***  ,   ***CD48***  , CD58 and   ***CD80***  . The reduction was
  not due to isotype switching to IgA, IgG or IgE. The role of   ***CD40***  ,
  CD48, CD58 and CD80 was further investigated after depletion
  of different cell types. Depletion of monocytes and NK cells resulted in
  no   ***detectable***   IgM production irrespective of added mAbs. In
  contrast, IgM production was still present after depletion of T cells and
  NK cells. Only mAb against CD80 and CD48 significantly reduced IgM
  production, the reduction of IgM production by anti-CD40 mAb was less
  than in the presence of T cells. Importantly, anti-CD58 mAb had no effect
  on IgM production after T cell and NK cell depletion. Taken together, the
  AM CD40, CD48, CD58, and CD80 are involved in Ig production of
  unseparated PBMCs. In this model of B cell differentiation only the AM
  CD58 depend on the presence of T cells while CD48 and CD80 help was found
  to be T cell independent.
? ds

Set     Items   Description
S1         14   E1-E3
S2          4   AU='ROTHE MARTIN'
S3        107   AU='THIEL ANDREAS'
S4          6   (S1 OR S2 OR S3) AND (CD40 OR CD40L RO CD154 OR CD40(W)LIG-
                 AND)
S5          3   RD S4  (unique items)
S6          0   (ANTI(W)CD40 OR CD40)(20N)(DETECT?)(CD4? OR CD8?)
S7      15412   (ANTI(W)CD40 OR CD40)(20N)(DETECT? OR ISOLAT? OR DETERMIN?
                 OR EXPRESS?)(20N)(CD4? OR CD8?)
S8       1098   (ANTI(W)CD40 OR CD40)(10N)(DETECT?)(10N)(CD4? OR CD8?)
S9        567   S8 AND PY<2002
S10       244   RD S9  (unique items)
? s (antibod?)(cd40L ro cd154 or cd40(W)ligand)(10n)(detect?)(10n)(cd4? or cd8?)
               0  ANTIBOD?)(CD40L RO CD154
           35372  CD40
               0  LIGAND)
         3779758  DETECT?
          384777  CD4?
          210228  CD8?
               0  CD40(W)LIGAND)(10N)DETECT?(10N)(CD4? OR CD8?)
     S11       0  (ANTIBOD?)(CD40L RO CD154 OR
                  CD40(W)LIGAND)(10N)(DETECT?)(10N)(CD4? OR CD8?)
? s (antibod?)(10n)(cd40L or cd154 or cd40(W)ligand)(10n)(detect?)(10n)(cd4? or
cd8?)
         2352215  ANTIBOD?
            8683  CD40L
            3845  CD154
           35372  CD40
          550185  LIGAND
           16639  CD40(W)LIGAND
         3779758  DETECT?
          384777  CD4?
          210228  CD8?
     S12     149  (ANTIBOD?)(10N)(CD40L OR CD154 OR
                  CD40(W)LIGAND)(10N)(DETECT?)(10N)(CD4? OR CD8?)
? rd s12
     S13      66  RD S12  (unique items)
? t s13/3/all
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13/3/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020241929 BIOSIS NO.: 200800288868
Human mesenchymal stem cells inhibit antibody production induced in vitro by allostimulation
AUTHOR: Comoli Patrizia (Reprint); Ginevri Fabrizio; Maccario Rita; Avanzini Maria Antonietta; Marconi Massimo; Groff Antonella; Cometa Angela; Cioni Michela; Porretti Laura; Barberi Walter; Frassoni Francesco ; Locatelli Franco
AUTHOR ADDRESS: Univ Pavia, Lab Sperimentale Trapianto Midollo Osseo, Fdn IRCCS Policlin S Matteo, Vle Golgi 19, I-27100 Pavia, Italy**Italy
AUTHOR E-MAIL ADDRESS: pcomoli@smatteo.pv.it
JOURNAL: Nephrology Dialysis Transplantation 23 (4): p1196-1202 APR 2008 2008
ITEM IDENTIFIER: doi:10.1093/ndt/gfm740
ISSN: 0931-0509
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020090518 BIOSIS NO.: 200800137457
Genninal center B cells are dispensable in prion transport and neuroinvasion
AUTHOR: Heikenwalder Mathias; Federaii Christian; Von Boehmer Lotta; Schwarz Petra; Wagner Mareike; Zeller Nicolas; Flaybaeck Johannes; Prinz Marco; Becher Burkhard; Aguzzi Adriano (Reprint)
AUTHOR ADDRESS: Univ Zurich Hosp, Inst Neuropathol, Schmelzbergstr 12, CH-8091 Zurich, Switzerland**Switzerland
AUTHOR E-MAIL ADDRESS: adriano@pathol.unizh.ch
JOURNAL: Journal of Neuroimmunology 192 (1-2): p113-123 DEC 2007 2007
ITEM IDENTIFIER: doi:10.1016/j.jneuroim.2007.09.022
ISSN: 0165-5728
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019901393 BIOSIS NO.: 200700561134
Identification and isolation of murine antigen-reactive T cells according to CD154 expression
AUTHOR: Kirchhoff Dennis; Frentsch Marco; Leclerk Patrick; Bumann Dirk; Rausch Sebastian; Hartmann Susanne; Thiel Andreas; Scheffold Alexander (Reprint)
AUTHOR ADDRESS: Deutsches Rheuma Forschungszentrum Berlin, Immunomodulat Grp, Charitepl 1, D-10117 Berlin, Germany**Germany
AUTHOR E-MAIL ADDRESS: scheffold@drfz.de
JOURNAL: European Journal of Immunology 37 (9): p2370-2377 SEP 2007 2007
ITEM IDENTIFIER: doi:10.1002/eji.200737322
ISSN: 0014-2980
DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: English

13/3/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019831601 BIOSIS NO.: 200700491342
Expression and function of the IL-2 receptor in activated human plasmacytoid dendritic cells
AUTHOR: Naranjo-Gomez Mar; Oliva Harold; Climent Nuria; Fernandez Marco A; Ruiz-Riol Marta; Bofill Margarita; Gatell Jose M; Gallart Teresa; Pujol-Borrell Ricardo; Borras Francesc E (Reprint)
AUTHOR ADDRESS: Inst Invest Germans Trias and Pujol, LIRAS, Banc Sang and Teixits, Ctra Can Ruti,Cami Escoles S-N, Badalona 08916, Barcelona, Spain **Spain
AUTHOR E-MAIL ADDRESS: feborras.liradbst.germanstrias@gencat.net
JOURNAL: European Journal of Immunology 37 (7): p1764-1772 JUL 2007 2007
ITEM IDENTIFIER: doi:10.1002/eji.200636980
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019607433 BIOSIS NO.: 200700267174
B cells play a cooperative role via CD40L-CD40 interaction in T cell-mediated experimental autoimmune neuritis in Lewis rats
AUTHOR: Zhu Wei; Mix Eilhard; Jin Tao; Adem Abdu; Zhu Jie (Reprint)
AUTHOR ADDRESS: Karolinska Univ Hosp Huddinge, Dept Neurobiol, Div Neurodegenerat and Neuroinflammat, Karolinska Inst, Novum,Plan 5, S-14186 Huddinge, Sweden**Sweden
AUTHOR E-MAIL ADDRESS: Jie.Zhu@ki.se
JOURNAL: Neurobiology of Disease 25 (3): p642-648 MAR 2007 2007
ISSN: 0969-9961
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19291027 BIOSIS NO.: 200600636422
Detection of memory B lymphocytes specific to hepatitis B virus (HBV) surface antigen (HBsAg) from HBsAg-vaccinated or HBV-immunized subjects by ELISPOT assay
AUTHOR: Tuaillon Edouard; Al Tabaa Yassine; Petitjean Gaeal; Huguet Marie-France; Pajeaux Georges; Fondere Jean-Michel; Ponseille Benoit; Ducos Jacques; Blanc Pierre; Vendrell Jean Pierre (Reprint)
AUTHOR ADDRESS: Inst Biol, Virol Lab, Av Doyen G Giraud, F-34295 Montpellier, France**France
AUTHOR E-MAIL ADDRESS: jp-vendrell@chu-montpellier.fr
JOURNAL: Journal of Immunological Methods 315 (1-2): p144-152 AUG 31 2006 2006

ISSN: 0022-1759
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19268506 BIOSIS NO.: 200600613901
Optimization of in vitro expansion of macaque CD4(+) T cells using anti-CD3 and co-stimulation for autotransfusion therapy
AUTHOR: Onlamoon Nattawat; Hudson Krystal; Bryan Patsy; Mayne Ann E; Bonyhadi Mark; Berenson Ron; Sundstrom Bruce J; Bostik Pavel; Ansari Aftab A; Villinger Francois (Reprint)
AUTHOR ADDRESS: Emory Univ, Dept Pathol and Lab Med, Sch Med, 101 Woodruff Circle Rm 2307, Atlanta, GA 30322 USA**USA
AUTHOR E-MAIL ADDRESS: fvillin@emory.edu
JOURNAL: Journal of Medical Primatology 35 (4-5): p178-193 AUG 2006 2006
ISSN: 0047-2565
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19058335 BIOSIS NO.: 200600403730
SLAM/SLAM interactions inhibit CD40-induced production of inflammatory cytokines in monocyte-derived dendritic cells
AUTHOR: Rethi Bence; Gogolak Peter; Szatmari Istvan; Veres Agota; Erdos Erika; Nagy Laszlo; Rajnavolgyi Eva; Terhorst Cox; Lanyi Arpad (Reprint)
AUTHOR ADDRESS: Debrecen Univ Med and Hlth Sci Ctr, Inst Immunol, 98 Nagyerdei Krt, H-4012 Debrecen, Hungary**Hungary
AUTHOR E-MAIL ADDRESS: alanyi@dote.hu
JOURNAL: Blood 107 (7): p2821-2829 APR 1 2006 2006
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18889331 BIOSIS NO.: 200600234726
Autoantibody to CD40 ligand in systemic lupus erythematosus: association with thrombocytopenia but not thromboembolism
AUTHOR: Nakamura M; Tanaka Y; Satoh T; Kawai M; Hirakata M; Kaburaki J; Kawakami Y; Ikeda Y; Kuwana M (Reprint)
AUTHOR ADDRESS: Keio Univ, Sch Med, Inst Adv Med Res, Shinjuku Ku, Tokyo 1608582, Japan**Japan
AUTHOR E-MAIL ADDRESS: kuwanam@sc.itc.keio.ac.jp
JOURNAL: Rheumatology (Oxford) 45 (2): p150-156 FEB 2006 2006
ISSN: 1462-0324
DOCUMENT TYPE: Article
RECORD TYPE: Abstract

LANGUAGE: English

13/3/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18835342 BIOSIS NO.: 200600180737
Differential expression and regulation of murine CD40 in regional vascular beds
AUTHOR: Vowinkel Thorsten; Wood Katherine C; Stokes Karen Y; Russell Janice ; Krieglstein Christian F; Granger D Neil (Reprint)
AUTHOR ADDRESS: Louisiana State Univ, Hlth Sci Ctr, Dept Mol and Cellular Physiol, 1501 Kings Hwy, Shreveport, LA 71130 USA**USA
AUTHOR E-MAIL ADDRESS: dgrang@lsuhsc.edu
JOURNAL: American Journal of Physiology - Heart and Circulatory Physiology 290 (2): pH631-H639 FEB 2006 2006
ISSN: 0363-6135
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/11 (Item 11 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18620080 BIOSIS NO.: 200510314580
Lymphocyte-associated LIGHT induces proinflammatory and prothrombotic changes in human vascular endothelial cells
AUTHOR: Celik Sultan (Reprint); Xia Ning; Gleissner Christian; Klingenberg Roland; Dengler Thomas J
AUTHOR ADDRESS: Univ Heidelberg, Heidelberg, Germany**Germany
JOURNAL: Circulation 110 (17, Suppl. S): p209 OCT 26 2004 2004
CONFERENCE/MEETING: 77th Scientific Meeting of the American-Heart-Association New Orleans, LA, USA November 07 -10, 2004; 20041107
SPONSOR: Amer Heart Assoc
ISSN: 0009-7322
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

13/3/12 (Item 12 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18599856 BIOSIS NO.: 200510294356
Functional characterization of antibodies against heparin-platelet factor 4 complex in heparin-induced thrombocytopenia patients in Asian-Indians: relevance to inflammatory markers
AUTHOR: Kannan Meganathan; Ahmad Sarfraz; Ahmad Firdos; Kale Shailaja; Hoppensteadt Debra A; Fareed Jawed; Saxena Renu (Reprint)
AUTHOR ADDRESS: All India Inst Med Sci, Dept Haematol, IRCH Bldg 1st Floor, New Delhi 110029, India**India
AUTHOR E-MAIL ADDRESS: renusax@hotmail.com
JOURNAL: Blood Coagulation & Fibrinolysis 16 (7): p487-490 OCT 2005 2005
ISSN: 0957-5235
DOCUMENT TYPE: Article
RECORD TYPE: Abstract

LANGUAGE: English

13/3/13 (Item 13 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18004431 BIOSIS NO.: 200400375220
The nonresponse to hepatitis B vaccination is associated with impaired lymphocyte activation
AUTHOR: Goncalves Loredana; Albarran Benibelks; Salmen Siham; Borges Lerida; Fields Howard; Montes Henry; Soyano Andres; Diaz Yuleima; Berrueta Lisbeth (Reprint)
AUTHOR ADDRESS: Inst Clin Immunol, Univ Los Andes, Ave 16 Septiembre,Edificio Louis Pasteur,Anexo 1A, Merida, 5101, Venezuela** Venezuela
AUTHOR E-MAIL ADDRESS: lberruet@ula.ve
JOURNAL: Virology 326 (1): p20-28 August 15, 2004 2004
MEDIUM: print
ISSN: 0042-6822 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/14 (Item 14 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17976645 BIOSIS NO.: 200400347434
Signalling via CD70, a member of the TNF family, regulates T cell functions
AUTHOR: Garcia Pilar; de Heredia Agustin Beltran; Bellon Teresa; Carpio Emilio; Llano Manuel; Caparros Esther; Aparicio Pedro; Lopez-Botet Miguel (Reprint)
AUTHOR ADDRESS: DCEXSMol Immunopathol Unit, Univ Pompeu Fabra, Dr Aiguader 80, E-08003, Barcelona, Spain**Spain
AUTHOR E-MAIL ADDRESS: miguel.lopez-botet@upf.edu
JOURNAL: Journal of Leukocyte Biology 76 (1): p263-270 July 2004 2004
MEDIUM: print
ISSN: 0741-5400 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/15 (Item 15 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17794364 BIOSIS NO.: 200400161705
Characterization of HIT-associated heparin-PF4 antibodies in Asian-Indians: Relevance to inflammatory markers.
AUTHOR: Kannan Meganathan (Reprint); Ahmad Sarfraz; Ahmed Rafeeq (Reprint); Kale Shailaja; Hoppensteadt Debra A; Fareed Jawed; Saxena Renu (Reprint)
AUTHOR ADDRESS: Haematology, All India Institute of Medical Sciences, New Delhi, India**India
JOURNAL: Blood 102 (11): p88b-89b November 16, 2003 2003
MEDIUM: print
CONFERENCE/MEETING: 45th Annual Meeting of the American Society of Hematology San Diego, CA, USA December 06-09, 2003; 20031206
SPONSOR: American Society of Hematology

ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

13/3/16 (Item 16 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17780676 BIOSIS NO.: 200400147337
Complexes of heparin and platelet factor 4 activate CD4 positive T cells from patients with heparin-induced thrombocytopenia (HIT).
AUTHOR: Banat G-Andre (Reprint); Hoppmann Sabine (Reprint); Ihlow Kerstin (Reprint); Matzdorff Axel; Pralle Hans (Reprint)
AUTHOR ADDRESS: Hematology, University of Giessen, Giessen, Germany** Germany
JOURNAL: Blood 102 (11): p549a November 16, 2003 2003
MEDIUM: print
CONFERENCE/MEETING: 45th Annual Meeting of the American Society of Hematology San Diego, CA, USA December 06-09, 2003; 20031206
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Poster; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

13/3/17 (Item 17 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17378886 BIOSIS NO.: 200300335629
Complexes of Heparin and Platelet Factor 4 Activate CD4 Positive T Cells from Patients with Heparin-Induced Thrombocytopenia (HIT).
AUTHOR: Hoppmann Sabine (Reprint); Ihlow Kerstin (Reprint); Usluoglu Nurguel (Reprint); Pralle Hans (Reprint); Banat Gamal-Andre (Reprint)
AUTHOR ADDRESS: Hematology and Oncology, University of Giessen, Giessen, Germany**Germany
JOURNAL: Blood 100 (11): pAbstract No. 1029 November 16, 2002 2002
MEDIUM: print
CONFERENCE/MEETING: 44th Annual Meeting of the American Society of Hematology Philadelphia, PA, USA December 06-10, 2002; 20021206
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Poster; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

13/3/18 (Item 18 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17366090 BIOSIS NO.: 200300324386
Lipoteichoic acid from Staphylococcus aureus enhances allergen-specific immunoglobulin E production in mice.
AUTHOR: Matsui K (Reprint); Nishikawa A
AUTHOR ADDRESS: Department of Immunobiology, Meiji Pharmaceutical University, 2-522-1 Noshio, Kiyose, Tokyo, 204-8588, Japan**Japan
AUTHOR E-MAIL ADDRESS: kmatsui@my-pharm.ac.jp

JOURNAL: Clinical and Experimental Allergy 33 (6): p842-848 June 2003 2003
MEDIUM: print
ISSN: 0954-7894 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/19 (Item 19 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16914279 BIOSIS NO.: 200200507790
The effects of malignant transformation on susceptibility of human urothelial cells to CD40-mediated apoptosis
AUTHOR: Bugajska Urszula; Georgopoulos Nikolaos T; Southgate Jennifer; Johnson Peter W M; Graber Pierre; Gordon John; Selby Peter J; Trejdosiewicz Ludwik K (Reprint)
AUTHOR ADDRESS: Lymphoepithelial Interactions Laboratory, Clinical Centre, Cancer Research U.K., St. James's University Hospital, Leeds, LS9 7TF, UK **UK
JOURNAL: Journal of the National Cancer Institute (Bethesda) 94 (18): p 1381-1395 September 18, 2002 2002
MEDIUM: print
ISSN: 0027-8874
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/20 (Item 20 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16900177 BIOSIS NO.: 200200493688
Allograft tolerance induced by intact active bone co-transplantation and anti-CD40L monoclonal antibody therapy
AUTHOR: Yin Dengping; Ma Lianli; Zeng Huasong; Shen Jikun; Chong Anita S (Reprint)
AUTHOR ADDRESS: Department of General Surgery, Rush Presbyterian St. Luke's Medical Center, 1653 W. Congress Parkway, Chicago, IL, 60612, USA**USA
JOURNAL: Transplantation (Baltimore) 74 (3): p345-354 August 15, 2002 2002
MEDIUM: print
ISSN: 0041-1337
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/21 (Item 21 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16765314 BIOSIS NO.: 200200358825
CD40 ligand (CD40L) does not stimulate proliferation of vascular smooth muscle cells
AUTHOR: Hermann Alexander; Schroer Karsten (Reprint); Weber Artur-Aron
AUTHOR ADDRESS: Institut fuer Pharmakologie und Klinische Pharmakologie, Heinrich-Heine-Universitaet, Moorenstr. 5, D-40225, Duesseldorf, Germany **Germany
JOURNAL: European Journal of Cell Biology 81 (4): p213-221 April, 2002

2002
MEDIUM: print
ISSN: 0171-9335
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/22 (Item 22 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16735774 BIOSIS NO.: 200200329285
Chronic lymphocytic leukemia B cells are endowed with the capacity to attract CD4+, CD40L+ T cells by producing CCL22
AUTHOR: Ghia Paolo; Strola Giuliana; Granziero Luisa; Geuna Massimo; Guida Giuseppe; Sallusto Federica; Ruffing Nancy; Montagna Licia; Piccoli Paola ; Chilosi Marco; Caligaris-Cappio Federico (Reprint)
AUTHOR ADDRESS: Laboratorio di Immunologia Oncologica, IRCC - Candiolo, Strada Provinciale 142, I-10060, Candiolo, Italy**Italy
JOURNAL: European Journal of Immunology 32 (5): p1403-1413 May, 2002 2002
MEDIUM: print
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/23 (Item 23 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16656578 BIOSIS NO.: 200200250089
LIGHT, a TNF family member enhances the antigen presenting capacity of chronic lymphocytic leukemia cells and stimulates autologous cytolytic T-cells
AUTHOR: Tolba Khaled A (Reprint); Bowers William J; Eling David; Casey Ann E; Kipps Thomas J; Federoff Howard J; Rosenblatt Joseph D
AUTHOR ADDRESS: James P Wilmot Cancer Center, University of Rochester, Rochester, NY, USA**USA
JOURNAL: Blood 98 (11 Part 1): p730a-731a November 16, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001; 20011207
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

13/3/24 (Item 24 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16543371 BIOSIS NO.: 200200136882
Blockade of CD40/CD40 ligand interactions prevents induction of factor VIII inhibitors in hemophilic mice but does not induce lasting immune tolerance
AUTHOR: Reipert Birgit M; Sasgary Maria; Ahmad Rafi U; Auer Wilfried; Turecek Peter L; Schwarz Hans Peter (Reprint)

AUTHOR ADDRESS: Baxter BioScience, Industriestrasse 67, A-1220, Vienna, Austria**Austria
JOURNAL: Thrombosis and Haemostasis 86 (6): p1345-1352 December, 2001 2001
MEDIUM: print
ISSN: 0340-6245
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/25 (Item 25 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16271915 BIOSIS NO.: 200100443754
High frequency of circulating HBcAg-specific CD8 T cells in hepatitis B infection: A flow cytometric analysis
AUTHOR: Matsumura S; Yamamoto K (Reprint); Shimada N; Okano N; Okamoto R; Suzuki T; Hakoda T; Mizuno M; Higashi T; Tsuji T
AUTHOR ADDRESS: First Department of Internal Medicine, Okayama University Medical School, 2-5-1, Shikata-cho, Okayama, 700-8558, Japan**Japan
JOURNAL: Clinical and Experimental Immunology 124 (3): p435-444 June, 2001 2001
MEDIUM: print
ISSN: 0009-9104
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/26 (Item 26 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16075323 BIOSIS NO.: 200100247162
Blocking the CD154-CD40 interaction with anti-CD154 antibody differentially regulates interleukin-4 synthesis in T cells and IgE production in B cells
AUTHOR: Koshio Takehiro; Kajiwara Keiichi; Ikizawa Koichi; Nakagami Keiji; Yanagihara Yukiyoshi (Reprint)
AUTHOR ADDRESS: Clinical Research Center for Allergy, National Sagamihara Hospital, 18-1 Sakuradai, Sagamihara, 228-8522, Japan**Japan
JOURNAL: Allergology International 50 (1): p35-41 2001 2001
MEDIUM: print
ISSN: 1323-8930
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/27 (Item 27 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15812229 BIOSIS NO.: 200000530542
Role of platelet P-selectin and CD40 ligand in the induction of monocytic tissue factor expression
AUTHOR: Lindmark Eva; Tenno Taavo; Siegbahn Agneta (Reprint)
AUTHOR ADDRESS: Department of Medical Sciences, Clinical Chemistry, University Hospital, S-75185, Uppsala, Sweden**Sweden
JOURNAL: Arteriosclerosis Thrombosis and Vascular Biology 20 (10): p

2322-2328 October, 2000 2000
MEDIUM: print
ISSN: 1079-5642
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/28 (Item 28 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15485538 BIOSIS NO.: 200000203851
High sequence homology between human and pig CD40 with conserved binding to human CD154
AUTHOR: Rushworth Stuart A; Bravery Christopher A; Thompson Simon (Reprint)
AUTHOR ADDRESS: Molecular Biology Unit, Imutran Limited (A Novartis Pharma AG Company), Cambridge, CB2 2YP, UK**UK
JOURNAL: Transplantation (Baltimore) 69 (5): p936-940 March 15, 2000 2000
MEDIUM: print
ISSN: 0041-1337
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/29 (Item 29 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15473528 BIOSIS NO.: 200000191841
The formation of immunogenic major histocompatibility complex class II-peptide ligands in lysosomal compartments of dendritic cells is regulated by inflammatory stimuli
AUTHOR: Inaba Kayo; Turley Shannon; Iyoda Tomonori; Yamaide Fumiya; Shimoyama Susumu; Reis e Sousa Caetano; Germain Ronald N; Mellman Ira; Steinman Ralph M (Reprint)
AUTHOR ADDRESS: Laboratory of Cell Physiology and Immunology, The Rockefeller University, 1230 York Ave., 405 Bronk Bldg., New York, NY, 10021-6399, USA**USA
JOURNAL: Journal of Experimental Medicine 191 (6): p927-936 March 20, 2000 2000
MEDIUM: print
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/30 (Item 30 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15321415 BIOSIS NO.: 200000039728
Expression of CD40 and its ligand, CD40L, in intestinal lesions of Crohn's disease
AUTHOR: Battaglia Edda; Biancone Luigi; Resegotti Andrea; Emanuelli Giorgio ; Fronda Gian Ruggero; Camussi Giovanni (Reprint)
AUTHOR ADDRESS: Cattedra di Nefrologia, Dipartimento di Medicina Interna, Universita di Torino, Corso Dogliotti 14, 10126, Torino, Italy**Italy
JOURNAL: American Journal of Gastroenterology 94 (11): p3279-3284 Nov.,

1999 1999
MEDIUM: print
ISSN: 0002-9270
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/31 (Item 31 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14642989 BIOSIS NO.: 199800437236
Therapy with antibodies against CD40L (CDI54) and CD44-variant isoforms reduces experimental autoimmune encephalomyelitis induced by a proeteolipid protein peptide
AUTHOR: Laman J D (Reprint); Maassen C B M; Schellekens M M; Visser L; Kap M; De Jong E; Van Puijenbroek M; Van Stipdonk M J B; Van Meurs M; Schwarzler C; Gunthert U
AUTHOR ADDRESS: Div. Immunological Infect. Dis., TNO Prevention Health, PO Box 2215, 2301 CE Leiden, Netherlands**Netherlands
JOURNAL: Multiple Sclerosis 4 (3): p147-153 June, 1998 1998
MEDIUM: print
ISSN: 1352-4585
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/32 (Item 32 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14434691 BIOSIS NO.: 199800228938
Chronic lymphocytic leukemia B cells can express CD40 ligand and demonstrate T-cell type costimulatory capacity
AUTHOR: Schattner Elaine J (Reprint); Mascarenhas John; Reyfman Inna; Koshy Mary; Woo Caroline; Friedman Steven M; Crow Mary K
AUTHOR ADDRESS: Room C-640, Cornell Univ. Med. Coll., 1300 York Ave., New York, NY 10021, USA**USA
JOURNAL: Blood 91 (8): p2689-2697 April 15, 1998 1998
MEDIUM: print
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/33 (Item 33 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14386663 BIOSIS NO.: 199800180910
Regulation of cytoplasmic, surface and soluble forms of CD40 ligand in mouse B cells
AUTHOR: Wykes Michelle; Poudrier Johanne; Lindstedt Ragnar; Gray David (Reprint)
AUTHOR ADDRESS: Dep. Immunol., Imperial Coll. Sch. Med., Hammersmith Hosp., Du Cane Rd., London W12 0NN, UK**UK
JOURNAL: European Journal of Immunology 28 (2): p548-559 Feb., 1998 1998
MEDIUM: print

ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/34 (Item 34 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13925376 BIOSIS NO.: 199799559436
Insert venom immunotherapy induces interleukin-10 production and a Th2-to-Th1 shift, and changes surface marker expression in venom-allergic subjects
AUTHOR: Bellinghausen Iris; Metz Gudrun; Enk Alexander H; Christmann Steffen; Knop Juergen; Saloga Joachim (Reprint)
AUTHOR ADDRESS: Univ.-Hautklinik, Langenbeckstr. 1, D-55131 Mainz, Germany **Germany
JOURNAL: European Journal of Immunology 27 (5): p1131-1139 1997 1997
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/35 (Item 35 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13701828 BIOSIS NO.: 199799335888
Induction of cognate and non-cognate T-cell help for B-cell IgE production in relation to CD40 ligand expression
AUTHOR: Armerding Dieter (Reprint); Hren Andrea; Callard Robin E; Fu Shu Man; Mudde Geert C
AUTHOR ADDRESS: Harvard Skin Disease Res. Cent., Div. Dermatol., Brigham and Woman's Hosp., Harvard Med. Sch., 75 Francis St., Boston, MA 02115, USA**USA
JOURNAL: International Archives of Allergy and Immunology 111 (4): p 376-384 1996 1996
ISSN: 1018-2438
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/36 (Item 36 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13448108 BIOSIS NO.: 199699082168
Human dendritic cells activate T lymphocytes via a CD40: CD40 ligand-dependent pathway
AUTHOR: McLellan Alexander D; Sorg Rudiger V; Williams Lisa A; Hart Derek N J (Reprint)
AUTHOR ADDRESS: Haematol./Immunol. Res. Group, Christchurch Hosp., P.O. Box 151, Christchurch, New Zealand**New Zealand
JOURNAL: European Journal of Immunology 26 (6): p1204-1210 1996 1996
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/37 (Item 37 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13181573 BIOSIS NO.: 199698649406
CD40 and B cell antigen receptor dual triggering of resting B lymphocytes turns on a partial germinal center phenotype
AUTHOR: Galibert Laurent (Reprint); Burdin Nicolas; De Saint-Vis Blandine; Garrone Pierre; Van Kooten Cees; Banchereau Jacques; Rousset Francoise
AUTHOR ADDRESS: Lab. Immunol. Res., Schering-Plough, 27 Chemin des Peupliers, B.P. 11, 69571 Dardilly Cedex, France**France
JOURNAL: Journal of Experimental Medicine 183 (1): p77-85 1996 1996
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/38 (Item 38 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13181555 BIOSIS NO.: 199698649388
Acquisition of CD40 expression during murine B-cell differentiation
AUTHOR: Grandien A (Reprint); Bras A; Martinez-A C
AUTHOR ADDRESS: Lab. 115 CSIC, Cent. Nacional Biotecnol., Consejo Superior Investigaciones Cientificas, Madrid, Spain**Spain
JOURNAL: Scandinavian Journal of Immunology 43 (1): p47-55 1996 1996
ISSN: 0300-9475
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/39 (Item 39 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13120661 BIOSIS NO.: 199698588494
Rapid induction of a novel costimulatory activity on B cells by CD40 ligand
AUTHOR: Wu Yan; Xu Jianchao; Shinde Sanjay; Grewal Iqbal; Henderson Tanya; Flavell Richard A (Reprint); Liu Yang
AUTHOR ADDRESS: Section Immunobiol., Howard Hughes Med. Inst., Yale Univ. Sch. Med., New Haven, CT 06510, USA**USA
JOURNAL: Current Biology 5 (11): p1303-1311 1995 1995
ISSN: 0960-9822
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/40 (Item 40 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13051690 BIOSIS NO.: 199598519523
CD40 ligand is constitutively expressed in a subset of T cell lymphomas and on the microenvironmental reactive T cells of follicular lymphomas and Hodgkin's disease

AUTHOR: Carbone Antonino (Reprint); Gloghini Annunziata; Gruss Hans-Jurgen; Pinto Antonio
AUTHOR ADDRESS: Div. Pathol., Centro Regionale Riferimento Oncol., IRCCS, via Pedemontana Occidentale, Aviano I-33081, Italy**Italy
JOURNAL: American Journal of Pathology 147 (4): p912-922 1995 1995
ISSN: 0002-9440
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/41 (Item 41 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12799797 BIOSIS NO.: 199598267630
A subset of CD4+ memory T cells contains preformed CD40 ligand that is rapidly but transiently expressed on their surface after activation through the T cell receptor complex
AUTHOR: Casamayor-Palleja Montserrat; Khan Mahmood; Maclennan Ian C M (Reprint)
AUTHOR ADDRESS: Dep. Immunol., Univ. Birmingham Med. Sch., Birmingham B15 2TT, UK**UK
JOURNAL: Journal of Experimental Medicine 181 (4): p1293-1301 1995 1995
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/42 (Item 42 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12719124 BIOSIS NO.: 199598186957
Gamma/delta T Lymphocytes Express CD40 Ligand and Induce Isotype Switching in B Lymphocytes
AUTHOR: Horner Anthony A; Jabara Haifa; Ramesh Narayanaswamy; Geha Raif S (Reprint)
AUTHOR ADDRESS: Div. Immunol., Children's Hosp., Dep. Pediatr., Harvard Med. Sch., 300 Longwood Ave., Boston, MA 02115, USA**USA
JOURNAL: Journal of Experimental Medicine 181 (3): p1239-1244 1995 1995
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/43 (Item 43 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12671998 BIOSIS NO.: 199598139831
A cytotoxic CD40/p55 tumor necrosis factor receptor hybrid detects CD40 ligand on herpesvirus saimiri-transformed T cells
AUTHOR: Hess Sigrun; Kurrle Roland; Lauffer Leander; Riethmueller Gert; Engelmann Hartmut (Reprint)
AUTHOR ADDRESS: Inst. Immunol., Goethestr. 31, D-80336 Muenchen, Germany** Germany
JOURNAL: European Journal of Immunology 25 (1): p80-86 1995 1995
ISSN: 0014-2980

DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/44 (Item 44 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12428391 BIOSIS NO.: 199497449676
Decreased expression of the ligand for CD40 in newborn lymphocytes
AUTHOR: Fuleihan Ramsay (Reprint); Ahern Deborah; Geha Raif S
AUTHOR ADDRESS: Div. Immunol., Enders 8, Children's Hosp., 300 Longwood Ave., Boston, MA 02115, USA**USA
JOURNAL: European Journal of Immunology 24 (8): p1925-1928 1994 1994
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

13/3/45 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

0082512259 EMBASE No: 2008320396
CD4 SUP +T cells in CIKs (CD4 SUP + CIKs) reversed resistance to fas-mediated apoptosis through CD40/CD40L ligation rather than IFN-gamma stimulation
Yu J.; Zhang W.; Jiang H.; Li H.; Cao S.; Ren X.
Department of Immunology, Tianjin Cancer Institute and Hospital, Tianjin Medical University, Tianjin, China
AUTHOR EMAIL: rwziyi@yahoo.com
CORRESP. AUTHOR/AFFIL: Ren X.: Department of Immunology, Tianjin Cancer Institute and Hospital, Huanhuxi Road, Tiyuanbei, Hexi District, Tianjin, China
CORRESP. AUTHOR EMAIL: rwziyi@yahoo.com

Cancer Biotherapy and Radiopharmaceuticals (Cancer Biother. Radiopharm.) (United States) June 1, 2008, 23/3 (342-354)
CODEN: CBRAF ISSN: 1084-9785
DOI: 10.1089/cbr.2007.0454
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 28

13/3/46 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

0082178743 EMBASE No: 2007592369
Germinal center B cells are dispensable in prion transport and neuroinvasion
Heikenwalder M.; Federau C.; Boehmer L.v.; Schwarz P.; Wagner M.; Zeller N.; Haybaeck J.; Prinz M.; Becher B.; Aguzzi A.
Institute of Neuropathology, University Hospital of Zurich, Schmelzbergstrasse 12, CH-8091 Zurich, Switzerland
AUTHOR EMAIL: adriano@pathol.unizh.ch
CORRESP. AUTHOR/AFFIL: Aguzzi A.: Institute of Neuropathology, University Hospital of Zurich, Schmelzbergstrasse 12, CH-8091 Zurich, Switzerland

CORRESP. AUTHOR EMAIL: adriano@pathol.unizh.ch

Journal of Neuroimmunology (J. Neuroimmunol.) (Netherlands) December 1, 2007, 192/1-2 (113-123)
CODEN: JNRID ISSN: 0165-5728
PUBLISHER ITEM IDENTIFIER: S016557280700327X
DOI: 10.1016/j.jneuroim.2007.09.022
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 54

13/3/47 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

0081063500 EMBASE No: 2006123577
The CD40/CD40 ligand system is expressed in the cutaneous lesions of erythema multiforme and Stevens-Johnson syndrome/toxic epidermal necrolysis spectrum
Caproni M.; Torchia D.; Schincaglia E.; Volpi W.; Frezzolini A.; Schena D.; Marzano A.; Quaglino P.; De Simone C.; Parodi A.; Barletta E.; Fabbri P.
Department of Dermatological Sciences, University of Florence, Via della Pergola 58/60, 50121 Florence, Italy
AUTHOR EMAIL: fabbri@unifi.it
CORRESP. AUTHOR/AFFIL: Fabbri P.: Department of Dermatological Sciences, University of Florence, Via della Pergola 58/60, 50121 Florence, Italy
CORRESP. AUTHOR EMAIL: fabbri@unifi.it

British Journal of Dermatology (Br. J. Dermatol.) (United Kingdom) February 1, 2006, 154/2 (319-324)
CODEN: BJDEA ISSN: 0007-0963 eISSN: 1365-2133
DOI: 10.1111/j.1365-2133.2005.07023.x
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 36

13/3/48 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

0080393439 EMBASE No: 2005037586
The role of CD40-CD154 interactions in autoimmunity and the benefit of disrupting this pathway
Toubi E.; Shoenfeld Y.
Div. of Allergy/Clinical Immunology, Bnai Zion Medical Center, Haifa, Israel; Technion Faculty of Medicine, Haifa, Israel
CORRESP. AUTHOR/AFFIL: Shoenfeld Y.: Department of Internal Medicine B, Center of Autoimmune Diseases, Sheba Medical Center, Tel Hashomer, Israel

Autoimmunity (Autoimmunity) (United Kingdom) September 1, 2004, 37/6-7 (457-464)
CODEN: AUIME ISSN: 0891-6934
DOI: 10.1080/08916930400002386
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 53

13/3/49 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

0080014361 EMBASE No: 2004199525
Coexpression of CD40 and CD40L on B lymphoma and carcinoma cells: An autocrine anti-apoptotic role
Voorzanger-Rousselot N.; Blay J.-Y.
Equipe Cytokines et Cancer, Unite INSERM U453, Centre Leon Berard, 28 rue Laennec, 69373 Lyon Cedex 08, France
AUTHOR EMAIL: blay@lyon.fnclcc.fr
CORRESP. AUTHOR/AFFIL: Blay J.-Y.: Equipe Cytokines et Cancer, Unite INSERM U453, Centre Leon Berard, 28 rue Laennec, 69373 Lyon Cedex 08, France
CORRESP. AUTHOR EMAIL: blay@lyon.fnclcc.fr

Leukemia and Lymphoma (Leuk. Lymphoma) (United Kingdom) June 1, 2004, 45/6 (1239-1245)
CODEN: LELYE ISSN: 1042-8194
DOI: 10.1080/1042819032000159834
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 34

13/3/50 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE

0079624502 EMBASE No: 2003332562
Human autologous dendritic cell-glioma fusions: Feasibility and capacity to stimulate T cells with proliferative and cytolytic activity
Sloan A.E.; Parajuli P.
Department of Neurosurgery, Wayne State University, Barbara Ann Karmanos Cancer Inst., 4160 John R. Road, Detroit, MI 48201, United States
AUTHOR EMAIL: asloan@neurosurgery.wayne.edu
CORRESP. AUTHOR/AFFIL: Sloan A.E.: Department of Neurosurgery, Wayne State University, Barbara Ann Karmanos Cancer Inst., 4160 John R. Road, Detroit, MI 48201, United States
CORRESP. AUTHOR EMAIL: asloan@neurosurgery.wayne.edu

Journal of Neuro-Oncology (J. Neuro-Oncol.) (United States) August 1, 2003, 64/1-2 (177-183)
CODEN: JNODD ISSN: 0167-594X
DOI: 10.1023/A:1024999707415
DOCUMENT TYPE: Journal; Conference Paper RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 31

13/3/51 (Item 7 from file: 73)
DIALOG(R)File 73:EMBASE

0079258619 EMBASE No: 2002422927
CD40 activation as potential tool in malignant neoplasms
Ottaiano A.; Pisano C.; De Chiara A.; Ascierto P.A.; Botti G.; Barletta E.; Apice G.; Gridelli C.; Iaffaioli V.R.
Division of Medical Oncology B, National Cancer Institute, Via Mariano Semmola, 80131 Naples, Italy
CORRESP. AUTHOR/AFFIL: Ottaiano A.: Division of Medical Oncology B,

National Cancer Institute, Via Mariano Semmola, 80131 Naples, Italy
CORRESP. AUTHOR EMAIL: ale.otto@libero.it

Tumori (Tumori) (Italy) September 1, 2002, 88/5 (361-366)
CODEN: TUMOA ISSN: 0300-8916
DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 82

13/3/52 (Item 8 from file: 73)
DIALOG(R)File 73:EMBASE

0078562743 EMBASE No: 2001168884
CD40 ligand expression in Mycobacterium bovis BCG infection and its regulation by cytokines: A direct role of interleukin 12
Mendez-Samperio P.; Garcia-Martinez E.
Departamento de Inmunologia, Escuela Nacional Ciencias Biologicas, Instituto Politecnico Nacional, 11340 Mexico, D.F., Mexico
CORRESP. AUTHOR/AFFIL: Mendez-Samperio P.: Departamento de Inmunologia, Escuela Nacional Ciencias Biologicas, Instituto Politecnico Nacional, 11340 Mexico, D.F., Mexico
CORRESP. AUTHOR EMAIL: pmendez@bios.encb.i.p.n.mx

Archives of Medical Research (Arch. Med. Res.) (United States) May 22, 2001, 32/2 (108-112)
CODEN: AEDEE ISSN: 0188-4409
PUBLISHER ITEM IDENTIFIER: S0188440901002582
DOI: 10.1016/S0188-4409(01)00258-2
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 19

13/3/53 (Item 9 from file: 73)
DIALOG(R)File 73:EMBASE

0078517595 EMBASE No: 2001123685
Analysis of the CD40/CD40L role in the sustenance of alloreactive antibody production
Shoker A.S.; Lun Z.-R.; Choudry R.; Saxena A.
Department of Medicine, University of Saskatchewan, Saskatoon, Sask. S7N OW8, Canada; Departments of Pathology, University of Saskatchewan, Saskatoon, Sask. S7N OW8, Canada; Division of Nephrology, Department of Medicine, Royal University Hospital, 103 Hospital Drive, Saskatoon, Sask. S7N OW8, Canada
AUTHOR EMAIL: shoker@sask.usask.ca
CORRESP. AUTHOR/AFFIL: Shoker A.S.: Division of Nephrology, Department of Medicine, Royal University Hospital, 103 Hospital Drive, Saskatoon, Sask. S7N OW8, Canada
CORRESP. AUTHOR EMAIL: shoker@sask.usask.ca

Transplant Immunology (Transplant Immunol.) (United Kingdom) April 11, 2001, 8/4 (219-228)
CODEN: TRIME ISSN: 0966-3274
PUBLISHER ITEM IDENTIFIER: S0966327401000326
DOI: 10.1016/S0966-3274(01)00032-6
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English

NUMBER OF REFERENCES: 54

13/3/54 (Item 10 from file: 73)
DIALOG(R)File 73:EMBASE

0078283506 EMBASE No: 2000333096
Expression of CD40 and CD40 ligand in Bowen's disease and squamous cell carcinoma
Amo Y.; Ohta Y.; Hamada Y.; Tatsuta M.; Katsuoka K.
Department of Dermatology, Kitasato Univ. School of Medicine, 1-15-1 Kitasato, Sagamihara, Kanagawa 228-8555, Japan
CORRESP. AUTHOR/AFFIL: Amo Y.: Department of Dermatology, Kitasato Univ. School of Medicine, 1-15-1 Kitasato, Sagamihara, Kanagawa 228-8555, Japan
CORRESP. AUTHOR EMAIL: amo@med.kitasato-u.ac.jp

European Journal of Dermatology (Eur. J. Dermatol.) (France) October 4, 2000, 10/6 (439-442)
CODEN: EJDEE ISSN: 1167-1122
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 19

13/3/55 (Item 11 from file: 73)
DIALOG(R)File 73:EMBASE

0077542733 EMBASE No: 1999028864
CD40L (CD 154) expression in human liver allografts during chronic ductopenic rejection
Gaweco A.S.; Wiesner R.H.; Yong S.; Krom R.; Porayko M.; Chejfec G.; McClatchey K.D.; Van Thiel D.H.
Liver Transplant Program, Loyola Univ. Chicago Medical Center, 2160 South First Ave, Maywood, IL 60153, United States
CORRESP. AUTHOR/AFFIL: Gaweco A.S.: Liver Transplant Program, Loyola Univ. Chicago Medical Center, 2160 South First Ave, Maywood, IL 60153, United States
CORRESP. AUTHOR EMAIL: agaweco@luc.edu

Liver Transplantation and Surgery (Liver Transplant. Surg.) (United States) February 22, 1999, 5/1 (1-7)
CODEN: LTSUF ISSN: 1074-3022
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 42

13/3/56 (Item 12 from file: 73)
DIALOG(R)File 73:EMBASE

0077391132 EMBASE No: 1998301549
Therapy with antibodies against CD40L (CD154) and CD44-variant isoforms reduces experimental autoimmune encephalomyelitis induced by a proteolipid protein peptide
Laman J.D.; Maassen C.B.M.; Schellekens M.M.; Visser L.; Kap M.; De Jong E.; Van Puijenbroek M.; Van Stipdonk M.J.B.; Van Meurs M.; Schwarzler C.; Gunthert U.
Div. Immunological and Infect. Dis., TNO Prevention and Health (TNO-PG),

PO Box 2215, 2301 CE Leiden, Netherlands; Department of Immunology, Erasmus University, Rotterdam, Netherlands
CORRESP. AUTHOR/AFFIL: Laman J.D.: Div. Immunological Infectious Dis., TNO Prevention and Health, PO Box 2215, 2301 CE Leiden, Netherlands

Multiple Sclerosis (Mult. Scler.) (United Kingdom) June 1, 1998, 4/3 (147-153)
CODEN: MUSCF ISSN: 1352-4585
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 25

13/3/57 (Item 13 from file: 73)
DIALOG(R)File 73:EMBASE

0076846513 EMBASE No: 1997139570
Insect venom immunotherapy induces interleukin-10 production and a Th2-to-Th1 shift, and changes surface marker expression in venom-allergic subjects
Bellinghausen I.; Metz G.; Enk A.H.; Christmann S.; Knop J.; Saloga J.
Clinical Research Group, Department of Dermatology, University of Mainz, Mainz, Germany
CORRESP. AUTHOR/AFFIL: Saloga J.: Universitats-Hautklinik, Langenbeckstr. 1, D-55131 Mainz, Germany

European Journal of Immunology (EUR. J. IMMUNOL.) (Germany) March 1, 1997, 27/5 (1131-1139)
CODEN: EJIMA ISSN: 0014-2980
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 52

13/3/58 (Item 14 from file: 73)
DIALOG(R)File 73:EMBASE

0076434870 EMBASE No: 1996111086
CD40 expression by human peripheral blood eosinophils
Ohkawara Y.; Lim K.G.; Xing Z.; Glibetic M.; Nakano K.; Dolovich J.; Croitoru K.; Weller P.F.; Jordana M.
Department of Pathology, McMaster University, Hamilton, Ont. L8N 3Z5, Canada
CORRESP. AUTHOR/AFFIL: Jordana M.: Department of Pathology, McMaster University, 1200 Main St. West, Hamilton, Ont. L8N 3Z5, Canada

Journal of Clinical Investigation (J. CLIN. INVEST.) (United States) April 1, 1996, 97/7 (1761-1766)
CODEN: JCINA ISSN: 0021-9738
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 33

13/3/59 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

17436617 PMID: 17406204

Live-cell assay to detect antigen-specific CD4+ T-cell responses by CD154 expression.
Chattopadhyay Pratip K; Yu Joanne; Roederer Mario
Immunotechnology Section, Vaccine Research Center, National Institute of Allergy and Infectious Diseases, National Institutes of Health, 40 Convent Drive, Bethesda, Maryland 20892, USA. pchattop@mail.nih.gov
Nature protocols (England) 2006, 1 (1) p1-6, ISSN 1750-2799--Electronic Journal Code: 101284307
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

13/3/60 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

147400906 CA: 147(19)400906c JOURNAL
Development and estimation of enzyme-linked immunosorbent assay kit for rapid detection of soluble CD40 ligand
AUTHOR(S): Bao, Pin-hong; Xu, Bang-lao; Bei, Chun-hua; Ma, Wei; Wang, Rong; Lei, Xiu-xia
LOCATION: Affiliated First People's Hospital, Guangzhou Medical College, Guangzhou, Peop. Rep. China, 510180
JOURNAL: Guangdong Yixue (Guangdong Yixue) DATE: 2007 VOLUME: 28
NUMBER: 2 PAGES: 187-189 CODEN: GUYIEG ISSN: 1001-9448 LANGUAGE: Chinese PUBLISHER: Guangdongsheng Yixue Qingbao Yanjiuso

13/3/61 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

146247111 CA: 146(13)247111x PATENT
Enzyme-linked immunosorbent assay test kit for early prediction of acute coronary syndrome through detecting soluble CD40 ligand
INVENTOR(AUTHOR): Xu, Banglao; Bei, Chunhua; Wang, Rong; Liu, Wanli; Zhang, Ge
LOCATION: Peop. Rep. China,
ASSIGNEE: Guangzhou First People's Hospital
PATENT: Faming Zh. Sh. Gong. Shuom ; CN 1904616 A DATE: 20070131
APPLICATION: CN 10036150 (20050728)
PAGES: 10pp. CODEN: CNXXEV LANGUAGE: Chinese
PATENT CLASSIFICATIONS:

IPCR/8 + Level	Value	Position	Status	Version	Action	Source	Office:
G01N-0033/577	A	I	F	B	20060101	H	CN
G01N-0033/543	A	I	L	B	20060101	H	CN
G01N-0033/531	A	I	L	B	20060101	H	CN

13/3/62 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

144449369 CA: 144(24)449369g PATENT
Detection of antibodies against calcium binding protein-hydroxy apatite complexes
INVENTOR(AUTHOR): Kajander, E. Olavi; Aho, K. M.; Ciftcioglu, Neva
LOCATION: USA

ASSIGNEE: Nanobac Life Sciences
PATENT: PCT International ; WO 200652924 A2 DATE: 20060518
APPLICATION: WO 2005US40358 (20051108) *US 2004PV625572 (20041108)
PAGES: 44 pp. CODEN: PIXXD2 LANGUAGE: English
PATENT CLASSIFICATIONS:
IPCR/8 + Level Value Position Status Version Action Source Office:
G01N-0033/543 A I F B 20060101 H EP
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BW; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; EG; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KM; KN; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; LY; MA; MD; MG; MK; MN; MW; MX; MZ; NA; NG; NI; NO; NZ; OM; PG; PH; PL; PT; RO; RU; SC; SD; SE; SG; SK; SL; SM; SY; TJ; TM; TN; TR; TT; TZ; UA; UG; US; UZ; VC; VN; YU; ZA DESIGNATED REGIONAL: AT; BE; BG; CH; CY; CZ; DE; DK; EE; ES; FI; FR; GB; GR; HU; IE; IS; IT; LT; LU; LV; MC; NL; PL; PT; RO; SE; SI; SK; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD; TG; BW; GH; GM; KE; LS; MW; MZ; NA; SD; SL; SZ; TZ; UG; ZM; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM

13/3/63 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

144210927 CA: 144(12)210927a JOURNAL
Detection of peripheral blood lymphocytes CD23, CD40L and serum IgE levels in asthmatic children
AUTHOR(S): Liu, Jin; Zhang, Yan; Gong, Hongyu; Wang, Huaili
LOCATION: Department of Pediatrics, Zhengzhou Maternal and Child Care Hospital, Zhengzhou, Peop. Rep. China, 450003
JOURNAL: Zhengzhou Daxue Xuebao, Yixueban (Zhengzhou Daxue Xuebao, Yixueban) DATE: 2005 VOLUME: 40 NUMBER: 1 PAGES: 126-127 CODEN: ZDXYBA
ISSN: 1671-6825 LANGUAGE: Chinese PUBLISHER: Zhengzhou Daxue Xuebao, Yixueban Bianjibu

13/3/64 (Item 5 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

142175388 CA: 142(10)175388k PATENT
Immunoassay of detecting CD4loCD40hi T cells for diagnosis of autoimmune diseases
INVENTOR(AUTHOR): Wagner, David H.
LOCATION: USA
PATENT: PCT International ; WO 200506949 A2 DATE: 20050127
APPLICATION: WO 2004US21646 (20040707) *US 2003PV484655 (20030707)
PAGES: 57 pp. CODEN: PIXXD2 LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: A61B-000/A
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BW; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; EG; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NA; NI; NO; NZ; OM; PG; PH; PL; PT; RO; RU; SC; SD; SE; SG; SK; SL; SY; TJ; TM; TN; TR; TT; TZ; UA; UG; US; UZ; VC; VN; YU; ZA; ZM; ZW DESIGNATED REGIONAL: BW; GH; GM; KE; LS; MW; MZ; NA; SD; SL; SZ; TZ; UG; ZM; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM; AT; BE; BG; CH; CY; CZ; DE; DK; EE; ES; FI; FR; GB; GR; HU; IE; IT; LU; MC; NL; PL; PT; RO; SE; SI; SK; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD; TG

13/3/65 (Item 6 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

141378832 CA: 141(23)378832c PATENT
Methods for detecting intracellular defensins in various leukocyte subpopulations
INVENTOR(AUTHOR): D'Costa, Sybil S.
LOCATION: USA
ASSIGNEE: Beckman Coulter, Inc.
PATENT: U.S. Pat. Appl. Publ. ; US 20040219612 A1 DATE: 20041104
APPLICATION: US 428870 (20030502)
PAGES: 15 pp. CODEN: USXXCO LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: 435007210; G01N-033/567A

13/3/66 (Item 7 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

126221182 CA: 126(17)221182h JOURNAL
Expression of trimeric CD40 ligand in Pichia pastoris: use of a rapid method to detect high-level expressing transformants
AUTHOR(S): McGrew, Jeffrey T.; Leiske, Dan; Dell, Brad; Klinke, Ralph; Krasts, Dace; Wee, SiowFong; Abbott, Nick; Armitage, Richard; Harrington, Kimberly
LOCATION: Department of Cell Sciences, Immunex Corporation, 51 University Street, Seattle, WA, 98101, USA
JOURNAL: Gene DATE: 1997 VOLUME: 187 NUMBER: 2 PAGES: 193-200
CODEN: GENED6 ISSN: 0378-1119 PUBLISHER ITEM IDENTIFIER: 0378-1119(96)00747-0 LANGUAGE: English PUBLISHER: Elsevier
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13/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020241929 BIOSIS NO.: 200800288868
Human mesenchymal stem cells inhibit antibody production induced in vitro by allostimulation
AUTHOR: Comoli Patrizia (Reprint); Ginevri Fabrizio; Maccario Rita; Avanzini Maria Antonietta; Marconi Massimo; Groff Antonella; Cometa Angela; Cioni Michela; Porretti Laura; Barberi Walter; Frassoni Francesco ; Locatelli Franco
AUTHOR ADDRESS: Univ Pavia, Lab Sperimentale Trapianto Midollo Osseo, Fdn IRCCS Policlin S Matteo, Vle Golgi 19, I-27100 Pavia, Italy**Italy
AUTHOR E-MAIL ADDRESS: pcomoli@smatteo.pv.it
JOURNAL: Nephrology Dialysis Transplantation 23 (4): p1196-1202 APR 2008 2008
ITEM IDENTIFIER: doi:10.1093/ndt/gfm740
ISSN: 0931-0509
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background. Antibodies directed against alloantigens are implicated in the pathogenesis of several immune reactions complicating transplantation, including humoral rejection after solid organ transplantation. Mesenchymal stem cells (MSCs) have immunomodulatory capacity, since in vivo they may prolong skin graft survival in the

animal model and can rescue patients with life-threatening graft-versus-host disease.Methods. To investigate whether MSCs exert an inhibitory effect on antibody production during allostimulation, we stimulated peripheral blood mononuclear cells, obtained from healthy controls or sensitized patients undergoing dialysis for end-stage renal failure, in mixed lymphocyte culture (MLC), and evaluated immunoglobulin production either in the absence or in the presence of third-party allogeneic MSCs. We also evaluated the effect of MSCs on B-cell allostimulation performed adding to MLC a polyclonal stimulus delivered by an agonist anti-CD40 monoclonal ***antibody*** .Results. We found that the addition of MSCs at the beginning of MLC considerably inhibited immunoglobulin production in standard MLC, irrespective of the MSC dose employed. Conversely, immunoglobulin secretion induced by direct ***CD40*** - ***CD40L*** binding was not significantly inhibited. Furthermore, we demonstrated, in one sensitized patient, that secretion of donor-specific anti-HLA class I antibodies detected both in baseline serum and in the supernatant of control MLC was inhibited by the addition of MSCs. Mechanistically, the addition of MSCs induced a striking decrease of IL-5 production in the cultures.Conclusions. Our findings suggest that third-party MSC are able to suppress allo-specific antibody production in vitro, and may therefore help overcome a positive cross-match in sensitized transplant recipients.

13/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020090518 BIOSIS NO.: 200800137457
Genninal center B cells are dispensable in prion transport and neuroinvasion
AUTHOR: Heikenwalder Mathias; Federaii Christian; Von Boehmer Lotta; Schwarz Petra; Wagner Mareike; Zeller Nicolas; Flaybaeck Johannes; Prinz Marco; Becher Burkhard; Aguzzi Adriano (Reprint)
AUTHOR ADDRESS: Univ Zurich Hosp, Inst Neuropathol, Schmelzbergstr 12, CH-8091 Zurich, Switzerland**Switzerland
AUTHOR E-MAIL ADDRESS: adriano@pathol.unizh.ch
JOURNAL: Journal of Neuroimmunology 192 (1-2): p113-123 DEC 2007 2007
ITEM IDENTIFIER: doi:10.1016/j.jneuroim.2007.09.022
ISSN: 0165-5728
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Transmissible spongiform encephalopathies (TSEs) are fatal neurodegenerative diseases of animals and humans. Many TSEs are initiated by prion replication in the lymphoreticular system (LRS). The cellular and molecular prerequisites for prion trafficking within the LRS are not fully understood. Here we have manipulated CD40 and its ligand to investigate whether genetic or pharmacological ablation of germinal center B cells (GCBs), which migrate into and out of germinal centers, influences prion pathogenesis. In contrast to previous reports, no alteration of prion pathogenesis was detected in mice lacking ***CD40L*** and in mice treated with anti- ***CD40L*** ***antibodies*** . These results suggest that GCBs alone do not impact peripheral splenic prion transport, replication efficiency, or neuroinvasion, and point to other mechanisms affecting prion transport from lymphoreticular sites of replication to the nervous system. (c) 2007 Elsevier B.V. All rights reserved.

13/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019901393 BIOSIS NO.: 200700561134
Identification and isolation of murine antigen-reactive T cells according to CD154 expression
AUTHOR: Kirchhoff Dennis; Frentsch Marco; Leclerk Patrick; Bumann Dirk; Rausch Sebastian; Hartmann Susanne; Thiel Andreas; Scheffold Alexander (Reprint)
AUTHOR ADDRESS: Deutsches Rheuma Forschungszentrum Berlin, Immunomodulat Grp, Charitepl 1, D-10117 Berlin, Germany**Germany
AUTHOR E-MAIL ADDRESS: scheffold@drfz.de
JOURNAL: European Journal of Immunology 37 (9): p2370-2377 SEP 2007 2007
ITEM IDENTIFIER: doi:10.1002/eji.200737322
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: T helper (Th) cells are central regulators of adaptive immune responses. However, the detection of the small number of Th cells specific for a particular antigen or pathogen is still a major challenge. CD154 was recently introduced as a marker for antigen-specific Th cells. To date, this technology was not applicable for mice - arguably the most important immunological model system. ***CD154*** is difficult to detect due to its rapid removal from the cell surface upon binding to ***CD40*** during antigen-specific activation by APC. We present an efficient strategy to block the degradation of murine CD154 by combined use of ***antibodies*** against ***CD40*** and ***CD154*** . This strategy makes CD154 easily accessible for surface staining, which allows isolation and expansion of rare antigen specific Tcells. Importantly, CD154 identified all specific Tcells in strongly Th1- or Th2-polarized immune responses against pathogens like Salmonella typhimurium and Heligmosomoides polygyrus, independent of their potential to produce cytokines. We demonstrate that CD154 can in fact be used as a reliable marker for antigen-specific CD4 T cells in mice, offering a unique option to analyze, isolate and rapidly expand the entire pool of Th-cells generated during a physiological T cell response in vivo.

13/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019831601 BIOSIS NO.: 200700491342
Expression and function of the IL-2 receptor in activated human plasmacytoid dendritic cells
AUTHOR: Naranjo-Gomez Mar; Oliva Harold; Climent Nuria; Fernandez Marco A; Ruiz-Riol Marta; Bofill Margarita; Gatell Jose M; Gallart Teresa; Pujol-Borrell Ricardo; Borras Francesc E (Reprint)
AUTHOR ADDRESS: Inst Invest Germans Trias and Pujol, LIRAS, Banc Sang and Teixits, Ctra Can Ruti,Cami Escoles S-N, Badalona 08916, Barcelona, Spain **Spain
AUTHOR E-MAIL ADDRESS: feborras.liradbst.germanstrias@gencat.net
JOURNAL: European Journal of Immunology 37 (7): p1764-1772 JUL 2007 2007
ITEM IDENTIFIER: doi:10.1002/eji.200636980
ISSN: 0014-2980
DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Human and mouse plasmacytoid dendritic cells (PDC) express IL-2 mRNA specifically upon TLR stimulation, but not under CD40L stimulation. Even though the expression of the IL-2R by PDC has been described, the functional implications of this expression remain unknown. Here, we investigated the expression and function of the IL-2R in activated human PDC. The IL-2R alpha chain, CD25, is expressed in both CpG- and ***CD40L*** -activated PDC. CD25 expression is a relatively rapid event, as the receptor was detected 6 h after the initial activation signal. Exogenous IL-2 added to ***CD40L*** -activated PDC increased the expression of CD25, enhanced the secretion of pro-inflammatory cytokines and promotes PDC survival. CpG-activated PDC cultured in the presence of IL-2R-blocking monoclonal antibodies showed a reduced expression of pro-inflammatory cytokines, especially TNF-alpha. This reduction was dose and time dependent, suggesting a regulatory role of IL-2 in TNF secretion that might occur at the post-transcriptional level. These results indicate that the expression of the IL-2R is relevant to human PDC activation, and that IL-2 maybe an important auto- and/or paracrine factor modulating the activation and survival of PDC. Finally, CD25 expression may be considered as a useful early activation marker for human PDC.

13/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019607433 BIOSIS NO.: 200700267174
B cells play a cooperative role via CD40L-CD40 interaction in T cell-mediated experimental autoimmune neuritis in Lewis rats
AUTHOR: Zhu Wei; Mix Eilhard; Jin Tao; Adem Abdu; Zhu Jie (Reprint)
AUTHOR ADDRESS: Karolinska Univ Hosp Huddinge, Dept Neurobiol, Div Neurodegenerat and Neuroinflammat, Karolinska Inst, Novum,Plan 5, S-14186 Huddinge, Sweden**Sweden
AUTHOR E-MAIL ADDRESS: Jie.Zhu@ki.se
JOURNAL: Neurobiology of Disease 25 (3): p642-648 MAR 2007 2007
ISSN: 0969-9961
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The expression of co-stimulatory molecules CD40 and CD40L was examined over the course of experimental autoimmune neuritis (EAN) induced in Lewis rats by immunization with bovine peripheral nerve myelin. In draining lymph nodes, highest level of CD40L expression was seen on day 7 post immunization (p.i.), i.e. before onset of clinical signs of EAN, while CD40 expression was increased on day 14 p.i., i.e. at peak of clinical disease. In contrast, both CD40 and CD40L expressing cells in sciatic nerves, a target organ of EAN, peaked on day 14 p.i., large numbers of both expressing cells were mainly detected on day 14-21 p.i. After co-culture with EAN rat B cells bearing ***CD40*** , PO peptide 180-199-specific T cell line cells exhibited a rapid downregulation of ***CD40L*** expression. Furthermore, FAN rats had enhanced PO peptide 180-199-specific antibody responses on day 74 p.i., which might have contributed to their aggravated EAN and further demonstrated the role of ***antibodies*** in EAN. The results indicate that CD40L-CD40 interactions are involved in the initiation of the antigen-specific T cell responses associated with the generation and development of EAN, and may mediate autoantibody production in EAN.

Evidently, B cells play a cooperative role via CD40L-CD40 interaction in T cell-mediated FAN of Lewis rats. (c) 2006 Elsevier Inc. All rights reserved.

13/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19291027 BIOSIS NO.: 200600636422
Detection of memory B lymphocytes specific to hepatitis B virus (HBV) surface antigen (HBsAg) from HBsAg-vaccinated or HBV-immunized subjects by ELISPOT assay
AUTHOR: Tuaillon Edouard; Al Tabaa Yassine; Petitjean Gaeal; Huguet Marie-France; Pajeaux Georges; Fondere Jean-Michel; Ponseille Benoit; Ducos Jacques; Blanc Pierre; Vendrell Jean Pierre (Reprint)
AUTHOR ADDRESS: Inst Biol, Virol Lab, Av Doyen G Giraud, F-34295 Montpellier, France**France
AUTHOR E-MAIL ADDRESS: jp-vendrell@chu-montpellier.fr
JOURNAL: Journal of Immunological Methods 315 (1-2): p144-152 AUG 31 2006 2006
ISSN: 0022-1759
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: To improve the investigation of the role of human memory B lymphocytes following hepatitis B virus (HBV) infection or vaccination, we developed a method to characterize circulating memory B cells specific to hepatitis B surface antigen (HBsAg). Our approach combined: (1) purification of CD19(+) cells, (2) CD40-CD40L polyclonal stimulation, and (3) enumeration of memory B cells differentiated into anti-HBs antibody (Ab)-secreting cells (HBs-SCs) by a HBs-ELISPOT assay. In this way, HBs-SCs were ***detected*** in 17 HBsAg-vaccinated and nine HBV-immunized subjects including four individuals with serum anti-HBs Ab levels < 10 mIU/ml, but not in six controls. IgG(+), IgA(+) plus IgM(+) HBs-SCs, representing 5-1736 cells/10(6) circulating B cells and 0.02-0.58% of total immunoglobulin-SCs generated by the B cell polyclonal stimulation, were counted by an Ig two-colour ELISPOT assay. In addition, anti-HBs Abs were found in 8/15 supernatants recovered from B cell cultures which contained HBs-SCs, suggesting that the HBs-ELISPOT assay is more reliable in tracking HBsAg-specific memory B cells than ELISA measurement of anti-HBs Abs secreted in supernatants. This new approach could be useful to explore the presence and the longevity of HBsAg-specific memory B cells in vaccinated and immunized subjects, in chronic HBV infection and after liver transplantation for HBV-related disease. (c) 2006 Elsevier B.V. All rights reserved.

13/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19268506 BIOSIS NO.: 200600613901
Optimization of in vitro expansion of macaque CD4(+) T cells using anti-CD3 and co-stimulation for autotransfusion therapy
AUTHOR: Onlamoon Nattawat; Hudson Krystal; Bryan Patsy; Mayne Ann E; Bonyhadi Mark; Berenson Ron; Sundstrom Bruce J; Bostik Pavel; Ansari Aftab A; Villinger Francois (Reprint)
AUTHOR ADDRESS: Emory Univ, Dept Pathol and Lab Med, Sch Med, 101 Woodruff Circle Rm 2307, Atlanta, GA 30322 USA**USA

AUTHOR E-MAIL ADDRESS: fvillin@emory.edu
JOURNAL: Journal of Medical Primatology 35 (4-5): p178-193 AUG 2006 2006
ISSN: 0047-2565
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background Our laboratory has previously shown that adoptive transfer of in vitro-expanded autologous purified polyclonal CD4(+) T cells using anti-CD3/CD28-coated beads induced antiviral responses capable of controlling SIV replication in vivo.Methods As CD4(+) T cells comprise several phenotypic and functional lineages, studies were carried out to optimize the in vitro culture conditions for maximal CD4(+) T-cell expansion, survival and delineate the phenotype of these expanded CD4(+) T cells to be linked to maximal clinical benefit.Results and Conclusions The results showed that whereas anti-monkey CD3 gamma/epsilon was able to induce T-cell proliferation and expansion in combination with antibodies against multiple co-stimulatory molecules, monkey CD3 epsilon cross reacting antibodies failed to induce proliferation of macaque ***CD4*** (+) T cells. Among co-stimulatory signals, anti-CD28 stimulation was consistently superior to anti-4-1BB, CD27 or ICOS while the use of anti-CD154 failed to deliver a detectable proliferation signal. Increasing the relative anti-CD28 co-stimulatory signal relative to anti-CD3 provided a modest enhancement of expansion. Additional strategies for optimization included attempts to neutralize free radicals, enhancement of glucose uptake by T cells or addition of T-cell stimulatory cytokines. However, none of these strategies provided any detectable proliferative advantage. Addition of 10 autologous irradiated feeder cells/expanding T cell provided some enhancement of expansion; however, given the high numbers of T cell needed, this approach was deemed impractical and costly, and lower ratios of feeder to expanding T cells failed to provide such benefit. The most critical parameter for efficient expansion of purified CD4(+) T cells from multiple monkeys was the optimization of space and culture conditions at culture inception. Finally, anti-CD3/28-expanded CD4(+) T cells uniformly exhibited a central memory phenotype, absence of CCR5 expression, marked CXCR4 expression in vitro, low levels of caspase 3 but also of Bcl-2 expression.

13/7/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19058335 BIOSIS NO.: 200600403730
SLAM/SLAM interactions inhibit CD40-induced production of inflammatory cytokines in monocyte-derived dendritic cells
AUTHOR: Rethi Bence; Gogolak Peter; Szatmari Istvan; Veres Agota; Erdos Erika; Nagy Laszlo; Rajnavolgyi Eva; Terhorst Cox; Lanyi Arpad (Reprint)
AUTHOR ADDRESS: Debrecen Univ Med and Hlth Sci Ctr, Inst Immunol, 98 Nagyerdei Krt, H-4012 Debrecen, Hungary**Hungary
AUTHOR E-MAIL ADDRESS: alanyi@dote.hu
JOURNAL: Blood 107 (7): p2821-2829 APR 1 2006 2006
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Signaling lymphocyte activation molecule (SLAM, CD150, or SLAMF1) is a self-ligand receptor on the surface of activated T- and B-lymphocytes, macrophages, and dendritic cells (DCs). Here we examine

the effect of SLAM/SLAM interactions on CD40L-induced CD40 signaling pathways in human DCs. CD40L-expressing L929 cells induced DCs to produce interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-alpha), and IL-12, which was strongly inhibited by coexpression of SLAM on the surface of the L929 cells. Similarly, transfection of DCs with SLAM strongly reduced CD40L-induced IL-12 production. Furthermore, the negative effect of SLAM/SLAM interactions on CD40L-induced DC activation was also detected in the presence of lipopolysaccharide (LPS). LPS-Induced IL-12 secretion, however, was not inhibited by SLAM engagement. CD40L-activated DCs affected by exposure to SLAM/SLAM engagement were impaired in their ability to induce differentiation of naive T lymphocytes into interferon-gamma (IFN-gamma)-producing T-helper 1 (Th1) effector cells. These inhibitory effects were not the result of a general unresponsiveness of DCs to CD40L, as SLAM/SLAM interactions did not prevent CD40L-induced up-regulation of CD83, CD86, or human leukocyte antigen (HLA)-DQ on the surface of DCs. Taken together, the results indicate that SLAM/SLAM interactions inhibit CD40 -induced signal transduction in monocyte-derived dendritic cells, an effect that was not detectable in earlier studies using anti-SLAM monoclonal ***antibodies*** .

13/7/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18889331 BIOSIS NO.: 200600234726
Autoantibody to CD40 ligand in systemic lupus erythematosus: association with thrombocytopenia but not thromboembolism
AUTHOR: Nakamura M; Tanaka Y; Satoh T; Kawai M; Hirakata M; Kaburaki J; Kawakami Y; Ikeda Y; Kuwana M (Reprint)
AUTHOR ADDRESS: Keio Univ, Sch Med, Inst Adv Med Res, Shinjuku Ku, Tokyo 1608582, Japan**Japan
AUTHOR E-MAIL ADDRESS: kuwanam@sc.itc.keio.ac.jp
JOURNAL: Rheumatology (Oxford) 45 (2): p150-156 FEB 2006 2006
ISSN: 1462-0324
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Objectives. To examine the prevalence, clinical associations and pathogenic roles of autoantibodies to CD40 ligand (CD40L) in patients with systemic lupus erythematosus (SLE).Methods. Plasma anti-CD40L antibodies from 125 patients with SLE, 24 with primary antiphospholipid syndrome (APS) and 90 with idiopathic thrombocytopenic purpura (ITP) and from 62 healthy individuals were measured with an enzyme-linked immunosorbent assay (ELISA). HeLa cells transfected with human CD40L cDNA (HeLa/CD40L) were used to confirm the presence of anti-CD40L autoantibodies. The effect of anti-CD40L antibodies on the CD40L-CD40 interaction was evaluated by observing CD40L-induced I kappa B activation in CD40-expressing fibroblasts.Results. Anti-CD40L autoantibody was detected in seven (6%) SLE, three (13%) primary APS and 11 (12%) ITP patients, but in no healthy controls. Antibody binding in an ELISA was competitively inhibited by membrane components of HeLa/CD40L. Anti-CD40L antibody-positive IgG specifically bound the surface of living HeLa/CD40L, as shown by flow cytometry. The frequency of thrombocytopenia was significantly higher in SLE patients with the anti-CD40L antibody than in those without (100 vs 14%; P < 0.00001), whereas there was no association between the anti-CD40L antibody and thrombosis. Binding of the anti-CD40L antibodies in patients' plasma to CD40L was competitively inhibited by a series of mouse anti-CD40L monoclonal

antibodies. Anti- ***CD40L*** antibody-positive IgG failed to inhibit ***CD40L*** -induced I kappa B activation.Conclusions. Anti- ***CD40L*** autoantibody is associated with thrombocytopenia but not thromboembolism. Our findings are potentially useful in understanding the complex roles of CD40L in the pathophysiology of thrombosis and haemostasis as well as the thromboembolic complications that occur during treatment with anti- ***CD40L*** humanized ***antibody*** .

13/7/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18835342 BIOSIS NO.: 200600180737
Differential expression and regulation of murine CD40 in regional vascular beds
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JOURNAL: American Journal of Physiology - Heart and Circulatory Physiology 290 (2): pH631-H639 FEB 2006 2006
ISSN: 0363-6135
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RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: There is emerging evidence for a role of the CD40/CD40 ligand (CD40L) dyad as a signaling mechanism in different inflammatory conditions. The aims of this study were to 1) quantify the constitutive and induced expression of CD40 in different regional vascular beds of the mouse and 2) assess the role of ***CD40L*** as a modulator of vascular endothelial ***CD40*** expression. The dual radiolabeled monoclonal antibody technique was used to quantify the expression of endothelial CD40 in control and LPS-challenged wild-type (WT) mice. Significant constitutive CD40 expression was detected in several vascular beds of WT mice with lung, kidney, and small intestine exhibiting the highest expression, whereas the liver and stomach showed no detectable baseline expression. LPS administration elicited two- to sevenfold increases in CD40 expression in several tissues (heart, kidney, and intestine) within 4 h, whereas other organs (brain) required up to 48 h to exhibit CD40 upregulation. CD40 expression was not detected in unstimulated or LPS-challenged CD40(-/-) mice. Constitutive expression of CD40 was profoundly reduced in unstimulated CD40L(-/-) mice, but the LPS-induced CD40 upregulation did not differ between CD40L(-/-) and WT mice. Depletion of platelets or T lymphocytes, the major CD40L-expressing cells in blood, also resulted in a profound reduction in basal CD40 expression. These findings demonstrate significant endothelial expression of CD40 under basal conditions in different vascular beds and increased CD40 expression after endothelial cell activation with LPS. Platelet- and T-lymphocyte-associated CD40L appears to play a major role in regulating the density of CD40 expression on vascular endothelial cells in vivo.

13/7/11 (Item 11 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18620080 BIOSIS NO.: 200510314580

Lymphocyte-associated LIGHT induces proinflammatory and prothrombotic changes in human vascular endothelial cells
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JOURNAL: Circulation 110 (17, Suppl. S): p209 OCT 26 2004 2004
CONFERENCE/MEETING: 77th Scientific Meeting of the American-Heart-Association New Orleans, LA, USA November 07 -10, 2004; 20041107
SPONSOR: Amer Heart Assoc
ISSN: 0009-7322
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The recently described tumor necrosis factor (TNF) family member LIGHT (herpes virus entry mediator ligand/TNFSF14), a ligand for the lymphotoxin beta receptor (LT beta R) and herpes virus entry mediator (TR2), is expressed on activated T lymphocytes and on various types of antigen presenting cells (e.g. dendritic cells). LIGHT has an important role in T cell costimulation and alloimmunity, the contribution of LIGHT and its receptors to endothelial cell activation and immunity is currently unknown. In the present study, LIGHT receptors TR2 and LT beta R were found to be expessed on native human vascular endothelial cells, expression of LIGHT itself could, however, not be demonstrated in native or cytokine-stimulated cells. In vitro stimulation of endothelial cells with recombinant soluble LIGHT resulted in dose-dependently increased expression of ICAM-1 (+ 350%), VCAM-1 (+ 250%), IL-8 (+ 400%) and tissue factor (+ 750%) on the mRNA and protein level, measured by RT-PCR, flow cytometry and ELISA, with a maximum at 50 ng/ml of LIGHT. Effects of LIGHT on endothelial cells appeared mediated by both types of receptors (TR2, LT beta R). Induction of gene expression by LIGHT stimulation was rapid with maximal increases of mRNA after 1-4 hours, dependent on NFkB activation and inhibitable by anti-LIGHT ***antibody*** . Moderately enhanced endothelial apoptosis was seen after LIGHT stimulation, potentiated by actinomycin D or cycloheximide, Endothelial activation by LIGHT was quantitatively comparable to soluble ***CD40*** ***ligand*** . Coculture of phytohemagglutinin (PHA)-activated lymphocytes similarly induced upregulation of ICAM-1, VCAM-1, IL-8 and tissue factor, which was inhibitable to > 50% by anti-LIGHT ***antibody*** . Soluble LIGHT was detected in cell-free supernatant of PHA-activated lymphocyte cultures, which induced similar endothelial stimulation as whole lymphocytes. In conclusion, membrane-bound and/or soluble forms of the novel costimulatory T lymphocyte antigen LIGHT induce pro-inflammatory and pro-thrombotic changes in human vascular endothelial cells independent of cognate antigen recognition, which appear quantitatively comparable to the well-characterized effects of CD40 ligand from the same TNF superfamily.

13/7/12 (Item 12 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18599856 BIOSIS NO.: 200510294356
Functional characterization of antibodies against heparin-platelet factor 4 complex in heparin-induced thrombocytopenia patients in Asian-Indians: relevance to inflammatory markers
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JOURNAL: Blood Coagulation & Fibrinolysis 16 (7): p487-490 OCT 2005 2005
ISSN: 0957-5235
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Occurrence of heparin-induced thrombocytopenia (HIT) was investigated for 33 Indian patients undergoing cardiovascular surgery who received unfractionated heparin (UFH). Platelet counts were performed prior to the initiation of UFH therapy and 5-16 days post therapy. Heparin-induced platelet aggregation, C-14-serotonin release assay, and enzyme-linked immunosorbent assay (ELISA) tests were performed in all the patients to detect the antibodies formed against the complex of heparin and platelet factor 4 (HPF4). Levels of inflammatory markers/mediators such as CD40 ligand (CD40L) and C-reactive protein (CRP) were also measured in the patient plasmas utilizing ELISA tests. Based on clinical observations and laboratory diagnoses, five patients (15%) were considered to have confirmed HIT. Despite wide variations in the titers of inflammatory markers, patients who tested ELISA-positive for HPF4 antibodies showed markedly elevated levels of both soluble CD40L and C-reactive protein. Most strikingly, the 14 C-serotonin release assay-positive patients showed up to a 10-fold increase in the level of CD40L. It is concluded that approximately 15% Asian-Indian patients receiving UFH during cardiovascular surgery develop functional HPF4 antibodies, which are associated with the increased levels of inflammatory markers/mediators in this catastrophic HIT syndrome.

13/7/13 (Item 13 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18004431 BIOSIS NO.: 200400375220
The nonresponse to hepatitis B vaccination is associated with impaired lymphocyte activation
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JOURNAL: Virology 326 (1): p20-28 August 15, 2004 2004
MEDIUM: print
ISSN: 0042-6822 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Nonresponsiveness against hepatitis B vaccination has been described in 4-10% of immunized subjects. We have explored the specific cell response to hepatitis B surface antigen by analyzing: PBMC proliferation, cytokine production (Th1, Th2 profiles, and TGF-beta), and activation molecules on Th cells. A poor proliferative response was demonstrated in nonresponders ($P < 0.05$). T cells from responders produced all tested cytokines ($P < 0.01$), in contrast with nonresponders subjects ($P < 0.05$). Expression of CD69 and CD25 was diminished in T cells from nonresponders ($P < 0.01$). A reduced expression of ***CD40L***

was also ***detected*** in T cells from nonresponders (P < 0.01). An elevated correlation coefficient was observed between CD40L on ***CD4*** + cells and ***antibody*** production. These results suggest an overall inability of T cells to be activated which could be consistent with potential differences in antigen presentation. In conclusion, our results suggest that an altered Th response may be a consequence of inappropriate early activation events. Copyright 2004 Elsevier Inc. All rights reserved.

13/7/14 (Item 14 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17976645 BIOSIS NO.: 200400347434
Signalling via CD70, a member of the TNF family, regulates T cell functions
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JOURNAL: Journal of Leukocyte Biology 76 (1): p263-270 July 2004 2004
MEDIUM: print
ISSN: 0741-5400 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: In the present work, we provide data supporting that CD70, a tumor necrosis factor (TNF)-related molecule, defined as the CD27 ligand (CD27L), may actively regulate T cell functions similarly to other members of the TNF family (i.e., ***CD40L*** and CD30L). Cross-linking CD70 with specific monoclonal antibodies (mAb) stimulated cytotoxicity and cytokine production in human T cell clones. Detection of intracellular-free calcium mobilization and mitogen-activated protein kinase phosphorylation upon mAb engagement of CD70 further supported an active signaling role for the TNF-related molecule. Similar results were obtained in the Jurkat leukaemia T cell line stably transfected with CD70; in that system, induction of Akt phosphorylation was detected, indirectly revealing the involvement of the phosphatidylinositol-3 kinase pathway. Stimulation of CD70+ Jurkat cells, with a CD70-specific mAb or with COS-7 cells transiently transfected with CD27, induced transcriptional activity detectable by different reporter gene expression systems. Altogether, our data point out that a reciprocal communication may be established between CD27+ and CD70+ cells during the immune response.

13/7/15 (Item 15 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17794364 BIOSIS NO.: 200400161705
Characterization of HIT-associated heparin-PF4 antibodies in Asian-Indians: Relevance to inflammatory markers.
AUTHOR: Kannan Meganathan (Reprint); Ahmad Sarfraz; Ahmed Rafeeq (Reprint); Kale Shailaja; Hoppensteadt Debra A; Fareed Jawed; Saxena Renu (Reprint)
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JOURNAL: Blood 102 (11): p88b-89b November 16, 2003 2003

MEDIUM: print
CONFERENCE/MEETING: 45th Annual Meeting of the American Society of Hematology San Diego, CA, USA December 06-09, 2003; 20031206
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Heparin-induced thrombocytopenia (HIT) is an under-recognized and potentially severe clinicopathologic complication of heparin therapy. The pathophysiology of HIT is complex due to the functional heterogeneity of heparin-platelet factor 4 (HPF4) antibodies, not all of which can activate platelets/endothelial cells. Thus, many patients develop HPF4 antibodies without clinical manifestation of HIT syndrome. To address this issue, immune-mediated type-II HIT was investigated in 33 Asian-Indian patients undergoing cardiovascular surgery who received unfractionated heparin (UFH), dosage of 4 mg/kg body weight (20,000 to 30,000 IU). The duration of the treatment varied from 5 to 16 days. Platelet counts were performed prior to the initiation of UFH therapy (baseline) and 5-16 days, post-therapy. The reduction in patients' platelet count (35-50%) of the baseline value or <100,000/mul was considered to be suggestive of HIT syndrome. Such laboratory tests of HIT diagnosis as heparin-induced platelet aggregation (HIPA), 14C-serotonin release assay (SRA), and enzyme-linked immunosorbent assay (ELISA), were performed in all the patients to detect the presence of HPF4 ***antibodies*** . HIT functional tests (HIPA and SRA) were performed exclusively by using platelets from HIT ***antibody*** -reactive donors. In addition, CD40 lagand (CD40L) and C-reactive protein (CRP) titers were also measured by using commercially available ELISA methods. Thrombocytopenia was found to be present in 10 (30%) patients. Of these, 4 patients were found to be positive by SRA test. One of these also had HIPA and ELISA positivity. An additional patient was tested to be positive by HIPA and ELISA. This patient had bleeding while the others were asymptomatic. Since the SRA and HIPA are considered to be highly sensitive/specific tests for functional HIT antibody detection, we determined an overall 5 (15%) HIT positivity with these tests. Amongst the 23 non-thrombocytopenic patients and controls, HIT antibodies were not detectable by HIPA test in all, but were detectable in 2 patients by ELISA and one patient by SRA. Since they did not have any clinical symptom including thrombocytopenia, it is possible that these 3 patients had either non-functional HIT antibodies or have antibodies directed against complexes formed between UFH and other chemokines of the C-X-C superfamily (such as IL-8 and/or NAP-2). A wide variation in the CD40L and CRP titers was observed in this group of samples. Patients who tested ELISA-positive for HPF4 ***antibodies*** , showed markedly elevated levels of both the ***CD40L*** and CRP (1.7 to 3.7-fold increase). Most strikingly, the SRA-positive patients showed up to 10-fold increase in the level of ***CD40L*** . Based on these observations, it is concluded that approximately 15% of the Indian patients receiving UFH during cardiovascular surgery develop functional HPF4 ***antibodies*** . Therefore, it is strongly recommended that in addition to clinical observations, heparin-treated patients should also be routinely monitored with the available laboratory assays for HIT diagnosis. Furthermore, the increased levels of both CD40L and CRP in these patients, especially those with positive HPF4 antibody liters, clearly suggest the role of inflammatory process in this catastrophic syndrome.

13/7/16 (Item 16 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

17780676 BIOSIS NO.: 200400147337
Complexes of heparin and platelet factor 4 activate CD4 positive T cells from patients with heparin-induced thrombocytopenia (HIT).
AUTHOR: Banat G-Andre (Reprint); Hoppmann Sabine (Reprint); Ihlow Kerstin (Reprint); Matzdorff Axel; Pralle Hans (Reprint)
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JOURNAL: Blood 102 (11): p549a November 16, 2003 2003
MEDIUM: print
CONFERENCE/MEETING: 45th Annual Meeting of the American Society of Hematology San Diego, CA, USA December 06-09, 2003; 20031206
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Poster; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Heparin-induced thrombocytopenia (HIT) is associated with antibodies specific for a complex of platelet factor 4 (PF4) and heparin. The role of anti-PF4/heparin antibodies in the pathogenesis of HIT have been shown frequently and lead after platelet activation to the generation of platelet-derived microparticles as procoagulent agents. Because of the participation of immunoglobulins, mostly IgG, we tested the hypothesis of T-cell involvement in this process. To characterize T-cell involvement peripheral blood mononuclear cells (PBMC) from five patients with classical HIT were obtained and restimulated with heparin/PF4 complexes, PF4 alone, heparin alone and medium alone in the presence or absence of autologous thrombocytes. Thereby we could detect a slightly proliferation of CD4+ T cells on day 4 of co-culture with autologous thrombocytes and the heparin/PF4 complexes whereas no proliferation of CD8+ T cells could be noted. Impressively we could observe a bright activation of this CD4+ T cells defined by an upregulation of the activation molecule CD25 as well as CD154. Suitable we could detect an upregulation of CD40 on the autologous B cells. In contrast to this culture, we couldn't observe any of these reactions by using PMCS of healthy donors. Because CD154 modulates physiologic processes, such as T cell mediated effector functions, general immune responses and triggers the expression of pro-inflammatory mediators, like cytokines and adhesion molecules associated with the pathogenesis of chronic inflammatory diseases, we postulate a CD4+ T cell involvement in the pathogenesis of HIT. To proof this hypothesis we extended our first assay by blocking CD 40L with a monoclonal antibody As a result we observed a disruption of the activation of ***CD4*** + cells. Moreover by preincubation of the autologous PBMC with anti-CD40L in the co-culture with thrombocytes and heparin/PF4 complex we could detect a significant decrease of the autoantibody-amount using the heparin/PF4 enzyme-linked immunoassay. Based on these results we test now the cell/cell interaction between B cells and CD4+ T cells through the CD40/CD40L system, which may possibly trigger and sustain the antibody production responsible for the pathogenesis of HIT.

13/7/17 (Item 17 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17378886 BIOSIS NO.: 200300335629
Complexes of Heparin and Platelet Factor 4 Activate CD4 Positive T Cells

from Patients with Heparin-Induced Thrombocytopenia (HIT).
AUTHOR: Hoppmann Sabine (Reprint); Ihlow Kerstin (Reprint); Usluoglu Nurguel (Reprint); Pralle Hans (Reprint); Banat Gamal-Andre (Reprint)
AUTHOR ADDRESS: Hematology and Oncology, University of Giessen, Giessen, Germany**Germany
JOURNAL: Blood 100 (11): pAbstract No. 1029 November 16, 2002 2002
MEDIUM: print
CONFERENCE/MEETING: 44th Annual Meeting of the American Society of Hematology Philadelphia, PA, USA December 06-10, 2002; 20021206
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Poster; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Heparin-induced thrombocytopenia (HIT) is associated with antibodies specific for a complex of platelet factor 4 (PF4) and heparin. The role of anti-PF4/heparin antibodies in the pathogenesis of HIT has been shown frequently and leads after platelet activation to the generation of platelet-derived microparticles as procoagulent agents. Because of the participation of immunoglobulins, mostly IgG, we tested the hypothesis of T-cell involvement in this process. To characterize T-cell involvement peripheral blood mononuclear cells (PBMC) from five patients with classical HIT were obtained and restimulated with heparin/PF4 complexes, PF4 alone, heparin alone and medium alone in the presence or absence of autologous thrombocytes. Thereby we could detect a slightly proliferation of CD4+ T cells on day 4 of co-culture with autologous thrombocytes and the heparin/PF4 complexes whereas no proliferation of CD8+ T cells could be noted. Impressively we could observe a bright activation of this CD4+ T cells defined by an upregulation of the activation molecule CD25 as well as CD154. Suitable we could detect an upregulation of CD40 on the autologous B cells. In contrast to this culture, we couldn't observe any of these reactions by using PMCS of healthy donors. Because CD154 modulates physiologic processes, such as T cell mediated effector functions, general immune responses and triggers the expression of pro-inflammatory mediators, like cytokines and adhesion molecules associated with the pathogenesis of chronic inflammatory diseases, we postulate an CD4+ T cell involvement in the pathogenesis of HIT. To proof this hypothesis we extended our first assay by blocking CD 40L with a monoclonal antibody As a result we observed a disruption of the activation of ***CD4*** + cells. Moreover by preincubation of the autologous PBMC with anti-CD40L in the co-culture with thrombocytes and heparin/PF4 complex we could detect a significant decrease of the autoantibody-amount using the heparin/PF4 enzyme-linked immunoassay. Assuming these results we conclude that the cell/cell interaction between B cells and CD4+ T cells through the CD40/CD40L system possibly triggers and sustain the antibody production responsible for the pathogenesis of HIT.

13/7/18 (Item 18 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17366090 BIOSIS NO.: 200300324386
Lipoteichoic acid from Staphylococcus aureus enhances allergen-specific immunoglobulin E production in mice.
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JOURNAL: Clinical and Experimental Allergy 33 (6): p842-848 June 2003 2003
MEDIUM: print
ISSN: 0954-7894 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background Our previous study demonstrated that lipoteichoic acid (LTA) from Staphylococcus aureus induced T helper type 2 (Th2)-prone dermatitis resembling that seen in atopic dermatitis (AD) patients in mice sensitized percutaneously with an allergen. However, the effects of LTA on allergen-specific IgE production in such sensitized mice have not been elucidated. Objective The purpose of this study was to determine the effects of LTA from S. aureus on allergen-specific IgE production in mice sensitized percutaneously with a house dust mite antigen (MA). Methods Mice were sensitized with a single topical application of MA and/or LTA to barrier-disrupted abdominal skin. One to 5 weeks later, MA-specific IgE antibodies in sera from sensitized mice were detected by an enzyme-linked immunosorbent assay (ELISA). Expression of B7.1 (***CD80***), B7.2 (***CD86***) and ***CD40L*** molecules by ***CD40***-positive (CD40+) and CD4-positive (CD4+) cells in the lymph nodes of sensitized mice were analysed by flow-cytometry (FACS). Results Simultaneous sensitization with MA and LTA increased IgE production 3 weeks later, significantly more than sensitization with MA alone. FACS analysis of CD40+ cells in the lymph nodes from sensitized mice showed that simultaneous sensitization with MA and LTA did not enhance CD80- or CD86-expression by antigen-presenting cells such as B lymphocytes and dendritic cells more than sensitization with MA alone. However, analysis of CD4+ cells in the lymph nodes showed that simultaneous sensitization with MA and LTA increased the number of CD40L-expressing Th cells more than sensitization with MA alone. Conclusion These results suggest that LTA enhances allergen-specific IgE production by a mechanism associated with up-regulation of CD40L-expressing Th cells and this might explain the role of skin colonization with S. aureus in AD patients.

13/7/19 (Item 19 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16914279 BIOSIS NO.: 200200507790
The effects of malignant transformation on susceptibility of human urothelial cells to CD40-mediated apoptosis
AUTHOR: Bugajska Urszula; Georgopoulos Nikolaos T; Southgate Jennifer; Johnson Peter W M; Graber Pierre; Gordon John; Selby Peter J; Trejdosiewicz Ludwik K (Reprint)
AUTHOR ADDRESS: Lymphoepithelial Interactions Laboratory, Clinical Centre, Cancer Research U.K., St. James's University Hospital, Leeds, LS9 7TF, UK **UK
JOURNAL: Journal of the National Cancer Institute (Bethesda) 94 (18): p 1381-1395 September 18, 2002 2002
MEDIUM: print
ISSN: 0027-8874
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: The tumor necrosis factor (TNF) superfamily of ligands and receptors mediates immune cell survival. Some members possess

a death domain, a protein motif that functions to transmit apoptotic signals, whereas others, such as CD40, do not. CD40 is expressed by both normal and malignant epithelial cells. To investigate the functional significance of this expression, we studied the effects of ligation of CD40, Fas, and TNF receptors (TNFRs) on the proliferation and survival of normal and malignant human urothelial cells and urothelial cells with disabled p53 function. Methods: Normal and malignant human urothelial cells were cultured with soluble TNF family agonists (CD40 ligand (CD40L), TNF-alpha, anti-Fas antibody, or cocultured with mouse fibroblasts stably transfected with plasmids that caused the cells to constitutively express CD40L or CD32; cell proliferation was estimated by an (3H)thymidine incorporation assay, and apoptosis was determined by Annexin V staining and by a DNA fragmentation assay. Messenger RNA levels for CD40 and potential downstream effector molecules were quantified by polymerase chain reaction-based and ribonuclease protection assays, respectively, and nuclear factor (NF) kappaB nuclear translocation was detected by immunofluorescence. All statistical tests were two-sided. Results: Soluble trimeric ***CD40L*** inhibited the growth of normal and malignant urothelial cells but did not induce apoptosis. Cell surface-presented ***CD40L*** induced massive apoptosis in CD40-positive transitional cell carcinoma cells but not in normal urothelial cells. Normal cells underwent ***CD40L*** -mediated apoptosis only in the presence of other TNFR agonists. An agonistic anti-CD40 antibody presented on the surface of CD32-transfected fibroblasts also induced apoptosis in transitional cell carcinoma cells and in normal urothelial cells. Apoptotic responses of tumor (but not normal) cells to soluble agonists were enhanced by blocking protein synthesis. Karyotypically normal urothelial cells with disabled p53 function underwent apoptosis during coculture with CD40L-expressing fibroblasts alone but were not additionally sensitive to additional TNFR agonists. Conclusions: Susceptibility to CD40 ligation-induced apoptosis may be a novel mechanism for eliminating neoplastically transformed urothelial cells. Loss of CD40 expression may be an important adaptive mechanism for transitional cell carcinoma development and progression.

13/7/20 (Item 20 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16900177 BIOSIS NO.: 200200493688
Allograft tolerance induced by intact active bone co-transplantation and anti-CD40L monoclonal antibody therapy
AUTHOR: Yin Dengping; Ma Lianli; Zeng Huasong; Shen Jikun; Chong Anita S (Reprint)
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JOURNAL: Transplantation (Baltimore) 74 (3): p345-354 August 15, 2002 2002
MEDIUM: print
ISSN: 0041-1337
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: One of the most promising approaches to achieving allograft tolerance involves the transient inhibition of co-stimulatory signals in T cells. There is, however, increasing evidence that this approach alone cannot universally elicit allograft tolerance and that adjunct therapies capable of synergizing with co-stimulation blockade may be necessary. Methods: We developed a novel tolerance strategy involving

co-transplantation of intact allogeneic bone fragments containing active bone marrow (intact active bone (IAB)) with heart allograft and transient anti- ***CD40L*** monoclonal ***antibody*** therapy. Results: Mice treated with IAB and anti-CD40L were tolerant to major histocompatibility complex and minor antigen-mismatched cardiac and skin allografts. Heart allografts had normal histology up to 270 days posttransplantation, and the production of graft-reactive ***antibodies*** was inhibited. Microchimerism, but no macrochimerism, of donor cells was detected in the peripheral blood or lymphoid organs of tolerant mice receiving IAB and anti- ***CD40L*** . Lymphocytes from tolerant mice retained normal proliferative responsiveness to donor cells in vitro but demonstrated a donor-specific loss in the priming of interferon-gamma responses. The ability to produce interleukin-2 or -4 when stimulated with donor cells was normal. Conclusions: Contrary to previous reports of the ability of bone marrow cells to induce central deletional tolerance, our data suggest that the regimen involving co-transplantation of IAB on the day of heart allograft transplantation and transient anti-CD40L therapy induces a robust donor-specific peripheral tolerance.

13/7/21 (Item 21 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16765314 BIOSIS NO.: 200200358825
CD40 ligand (CD40L) does not stimulate proliferation of vascular smooth muscle cells
AUTHOR: Hermann Alexander; Schroer Karsten (Reprint); Weber Artur-Aron
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JOURNAL: European Journal of Cell Biology 81 (4): p213-221 April, 2002
2002
MEDIUM: print
ISSN: 0171-9335
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The present study investigates the effects of CD40 ligand (CD40L) on mitogenic signalling, proliferation, and migration of cultured bovine coronary artery smooth muscle cells (SMC). A time- and concentration-dependent phosphorylation of the extracellular signal-regulated kinases-1/2 (ERK-1/2) and the mitogen-activated protein kinase p38 (p38-MAPK) was observed upon stimulation with soluble ***CD40L*** (sCD40L). This phosphorylation was inhibited by neutralizing ***antibodies*** against the ***CD40*** and ***CD40L*** , respectively. Activation of the phosphatidylinositol-3-phosphate (PI-3) kinase pathway by sCD40L, as determined by the measurement of Akt phosphorylation, was not ***detected*** . However, there was evidence for direct activation of the NFkappaB system (degradation of IkappaBalpha and nuclear translocation of the p65 NFkappaB subunit) by sCD40L. Accordingly, sCD40L caused a small but significant increase in DNA synthesis. However, sCD40L-induced DNA synthesis was not followed by proliferation (increase in cell number). Furthermore, sCD40L did not potentiate SMC mitogenesis induced by known mitogens such as platelet-derived growth factor-BB, thrombin or serum. The lack of cell proliferation was not caused by a concomitant induction of SMC apoptosis by sCD40L. The possible role of membrane-bound CD40L in SMC mitogenesis was also studied using different membrane preparations (platelets, lymphocytes). However, no mitogenic effects of membrane-bound CD40L were detected. Finally, sCD40L did not

induce SMC migration. From these data it is concluded that CD40L activates mitogenic signalling and DNA synthesis but does not contribute to proliferation or migration of vascular SMC.

13/7/22 (Item 22 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16735774 BIOSIS NO.: 200200329285
Chronic lymphocytic leukemia B cells are endowed with the capacity to attract CD4+, CD40L+ T cells by producing CCL22
AUTHOR: Ghia Paolo; Strola Giuliana; Granziero Luisa; Geuna Massimo; Guida Giuseppe; Sallusto Federica; Ruffing Nancy; Montagna Licia; Piccoli Paola ; Chilosi Marco; Caligaris-Cappio Federico (Reprint)
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JOURNAL: European Journal of Immunology 32 (5): p1403-1413 May, 2002 2002
MEDIUM: print
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The natural history of B-chronic lymphocytic leukemia (CLL) is not entirely explained by intrinsic defects of the neoplastic cell, but is also favored by microenvironmental signals. As CLL cells retain the capacity to respond to CD40 ligand (CD40L) and as CD4+ T cells are always present in involved tissues, we asked whether malignant CLL cells might produce T cell-attracting chemokines. We studied the chemokine expression of CD19+/CD5+ malignant B cells from peripheral blood (PB), lymph nodes (LN) or bone marrow (BM) of 32 patients and found a major difference. LN- and BM-, but not PB-derived cells, expressed a readily detectable reverse transcription-PCR band for CCL22 and one for CCL17 of variable intensity. ***CD40*** ligation of PB cells induced the mRNA expression of both CCL22 and CCL17. CCL22 was also released in the culture supernatants. These supernatants induced the migration of activated ***CD4*** +, ***CD40L*** + T cells expressing the CCL22 receptor, CCR4. T cell migration was abrogated by anti-CCL22 ***antibodies*** . Immunohistochemistry and cytofluorography studies revealed that a proportion of ***CD4*** + T cells in CLL LN and BM expressed ***CD40L*** . Our data demonstrate that malignant CLL cells chemo-attract CD4+ T cells that in turn induce a strong chemokine production by the leukemic clone, suggesting a vicious circle, leading to the progressive accumulation of the neoplastic cells.

13/7/23 (Item 23 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16656578 BIOSIS NO.: 200200250089
LIGHT, a TNF family member enhances the antigen presenting capacity of chronic lymphocytic leukemia cells and stimulates autologous cytolytic T-cells
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JOURNAL: Blood 98 (11 Part 1): p730a-731a November 16, 2001 2001
MEDIUM: print

CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001; 20011207
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Members of the TNF superfamily play a key role in immune regulation and activation of effector cells, an activity that has been extended to generate anti-tumor immune response both in vitro and in vivo, (CD40/CD40L, 4-1-BB/4-1BBL and OX-40/OX-40L). LIGHT, (TNFSF14) a recently cloned member of the TNF superfamily binds Hve-A (formerly known as HVEM), lymphotoxin beta receptor (LTbetaR) and DcR3/TR6. Signaling through the Hve-A receptor activates both the T-cell and antigen presenting cell (APC) through recruitment of members of the TRAF family of adaptor molecules that will eventually activate the NF-kappaB and AP-1 transcription factors, while binding the LTbetaR induces apoptosis in several tumor cell lines. The cDNA for human LIGHT was cloned by RT-PCR from dendritic cell mRNA, subcloned into an HSV amplicon plasmid and packaged using helper virus-free methodology. The packaged vector, hf-HSV-LIGHT, was used to transduce human chronic lymphocytic leukemia (CLL) cells. Expression of LIGHT on CLL cells was ***detected*** by flow-cytometry using both a soluble Hve-A/Fc fusion protein and anti-LIGHT monoclonal ***antibody*** . We studied the immune modulatory function and T-cell activation by LIGHT in comparison to CD40L, both delivered using helper-free HSV amplicon vectors. LIGHT expression induced up-regulation of B7.1, B7.2 and ICAM.1 on CLL cells, albeit to a lesser degree than seen in response to transduced CD40L. LIGHT expression enhanced antigen presenting capacity of the transduced CLL cells as shown in an allogeneic mixed lymphocyte tumor reaction (MLTR). Hf-HSV-LIGHT transduced CLL cells successfully stimulated the generation of specific autologous cytotoxic T-lymphocyte activity following in vitro priming. CTL activity was blocked by the anti-MHC-I antibody W6-32. Our data suggests that expression of LIGHT using an HSV amplicon vector may be a useful strategy for immune therapy of B-cell lymphoid malignancies and CLL gene therapy.

13/7/24 (Item 24 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16543371 BIOSIS NO.: 200200136882
Blockade of CD40/CD40 ligand interactions prevents induction of factor VIII inhibitors in hemophilic mice but does not induce lasting immune tolerance
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JOURNAL: Thrombosis and Haemostasis 86 (6): p1345-1352 December, 2001 2001
MEDIUM: print
ISSN: 0340-6245
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RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Patients with severe hemophilia A frequently develop neutralizing anti-factor VIII antibodies after replacement therapy with factor VIII (FVIII). In a search for new strategies to induce immune tolerance

against FVIII in these patients, we used a murine model of hemophilia A to investigate the importance of CD40/CD40 ligand (CD40L) interactions for the initiation of the anti-FVIII immune response. We focused our attention in particular on the induction of neutralizing anti-FVIII antibodies and the Th1/Th2 polarization of FVIII-specific T cells. The development of anti-FVIII antibodies was analyzed by ELISA systems (detection of total anti-FVIII antibodies) and Bethesda assays (determination of neutralizing anti-FVIII ***antibodies***). Factor VIII-specific T cells were characterized by multiparameter flow cytometry and cytokine ELISAs for the detection of cytokine production in splenic ***CD4*** +cntdotT cells after in vitro restimulation with FVIII. Hemophilic mice received four doses of FVIII and anti-CD40L ***antibody*** MR1 (24 h before FVIII). Subsequently mice received four doses of FVIII only. The induction of neutralizing anti-FVIII antibodies in hemophilic mice after treatment with human FVIII could be prevented completely by a blockade of CD40/CD40L interactions using MR1. Furthermore, FVIII-specific T-cell responses that included both Th1 and Th2 cells were suppressed when mice were treated with FVIII and MR1. The initial blockade of ***CD40*** / ***CD40L*** interactions was, however, not sufficient to induce a lasting immune tolerance against FVIII. The immune suppression was abolished and both neutralizing anti-FVIII antibodies and FVIII-specific T cells developed when treatment with FVIII was continued after the omission of MR1. In addition, there were no alterations in the Th1/Th2 polarization induced by the initial blockade of CD40/CD40L interactions.

13/7/25 (Item 25 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16271915 BIOSIS NO.: 200100443754
High frequency of circulating HBcAg-specific CD8 T cells in hepatitis B infection: A flow cytometric analysis
AUTHOR: Matsumura S; Yamamoto K (Reprint); Shimada N; Okano N; Okamoto R; Suzuki T; Hakoda T; Mizuno M; Higashi T; Tsuji T
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JOURNAL: Clinical and Experimental Immunology 124 (3): p435-444 June, 2001 2001
MEDIUM: print
ISSN: 0009-9104
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Viral antigen-specific T cells are important for virus elimination. We studied the hepatitis B virus (HBV)-specific T cell response using flow cytometry. Three phases of HBV infection were studied: Group A, HBeAg (+) chronic hepatitis; Group B, HBeAb (+) HBV carrier after seroconversion; and Group C, HBsAb (+) phase. Peripheral T cells were incubated with recombinant HB core antigen (HBcAg), and intracytoplasmic cytokines were analysed by flow cytometry. HBcAg-specific CD4 and CD8 T cells were identified in all three groups and the number of IFN-gamma-positive T cells was greater than TNF-alpha-positive T cells. The frequency of IFN-gamma-positive CD4 and CD8 T cells was highest in Group C, compared with Groups A and B. No significant difference in the HBcAg-specific T cell response was observed between Group A and Group B. The HBcAg-specific ***CD8*** T cell response was diminished by CD4 depletion, addition of antibody against human leucocyte antigen (HLA) class I, class II or ***CD40L*** .

Cytokine-positive CD8 T cells without HBcAg stimulation were present at a high frequency (7 of 13 cases) in Group B, but were rare in other groups. HBcAg-specific T cells can be ***detected*** at high frequency by a sensitive flow cytometric analysis, and these cells are important for controlling HBV replication.

13/7/26 (Item 26 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16075323 BIOSIS NO.: 200100247162
Blocking the CD154-CD40 interaction with anti-CD154 antibody differentially regulates interleukin-4 synthesis in T cells and IgE production in B cells
AUTHOR: Koshio Takehiro; Kajiwara Keiichi; Ikizawa Koichi; Nakagami Keiji; Yanagihara Yukiyoshi (Reprint)
AUTHOR ADDRESS: Clinical Research Center for Allergy, National Sagamihara Hospital, 18-1 Sakuradai, Sagamihara, 228-8522, Japan**Japan
JOURNAL: Allergology International 50 (1): p35-41 2001 2001
MEDIUM: print
ISSN: 1323-8930
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Using severe combined immunodeficiency mice engrafted with peripheral blood mononuclear cells from atopic patients, we analyzed the regulatory effects of anti-CD154 antibody on the in vivo induction of human interleukin (IL)-4 and IgE expression. Although anti-CD154 treatment of engrafted mice abrogated mature Cepsilon transcription and IgE production, IL-4 mRNA levels were enhanced by the treatment. In addition, anti-CD154-induced enhancement of intracellular IL-4 synthesis was detectable in both CD4+ and CD8+ T cell subsets. Furthermore, upregulation of germline Cepsilon transcription could be seen in anti- ***CD154*** -treated mice. These results demonstrate that blocking the CD154-CD40 interaction with anti-CD154 differentially regulates IL-4 synthesis in T cells and IgE production in B cells. Our data also indicate that ***antibody*** ligation of CD154 on T cells causes enhanced synthesis of IL-4, thereby contributing to upregulation of germline Cepsilon transcription in B cells.

13/7/27 (Item 27 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15812229 BIOSIS NO.: 200000530542
Role of platelet P-selectin and CD40 ligand in the induction of monocytic tissue factor expression
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JOURNAL: Arteriosclerosis Thrombosis and Vascular Biology 20 (10): p 2322-2328 October, 2000 2000
MEDIUM: print
ISSN: 1079-5642
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Activated platelets can express CD40 ligand (CD40L) and trigger inflammatory response and tissue factor (TF) expression in endothelial cells through interaction with CD40. This pathway is also important for T cell-induced monocyte and endothelial cell procoagulant activity. We have studied the potential role of the CD40-CD40L pathway in platelet-induced TF expression in a monocytic cell line and in whole-blood monocytes. In vitamin D3-differentiated U-937 cells, thrombin-stimulated platelets increased TF expression as measured by mRNA quantification, flow cytometry, and procoagulant activity. Maximum antigen expression occurred after 2 hours. Neutralizing anti-P-selectin ***antibody*** yielded a 50% suppression of procoagulant activity, whereas antibody to ***CD40L*** had no effect. In thrombin receptor activator-stimulated citrated blood, monocytes were up to 77% TF-positive, with peak expression after only 15 minutes. However, no TF mRNA was ***detectable*** at that time. Anti-P-selectin ***antibody*** reduced TF by 50%, whereas ***antibody*** to ***CD40L*** gave a 17% reduction. Thus, we conclude that P-selectin exposed on activated platelets induces the expression of TF in both U-937 cells and whole-blood monocytes but by different mechanisms. Platelet CD40L does not display any significant effect on U-937 cells but may be of some importance on whole-blood monocytes. This suggests a possible functional difference between U-937 and monocyte CD40. Another important finding in this study is the rapid appearance of surface TF on monocytes without detectable mRNA formation. This indicates that TF may be stored intracellularly in these cells and can be exposed on the surface independent of de novo protein synthesis.

13/7/28 (Item 28 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15485538 BIOSIS NO.: 200000203851
High sequence homology between human and pig CD40 with conserved binding to human CD154
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AUTHOR ADDRESS: Molecular Biology Unit, Imutran Limited (A Novartis Pharma AG Company), Cambridge, CB2 2YP, UK**UK
JOURNAL: Transplantation (Baltimore) 69 (5): p936-940 March 15, 2000 2000
MEDIUM: print
ISSN: 0041-1337
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: Understanding the molecular interactions between pig tissues and human immune cells is fundamental to achieving long-term pig to human xenograft survival. CD40 has been shown to be central in the interaction of T cells with many antigen-presenting cells including B cells, and dendritic cells. It has been clearly shown in vitro that human T cells can effectively recognize pig major histocompatibility complex proteins, and that various accessory molecule interactions are compatible between these species, including human CD28 with pig B7 family members (***CD80*** / ***CD86***). The importance of ***CD40*** in transplantation has been established using blocking antibodies to its ligand, CD154, which prolong allograft survival in mouse and primate models. Methods: Pig ***CD40*** was cloned from a porcine spleen cDNA library and subsequently sequenced. Expression of pig ***CD40*** was detected by flow cytometry using soluble human CD154 (hCD154-Ig). Results: Comparison of the derived amino acid sequence of

pig with human shows 74% identity. Significantly, there is conservation between pig and human at 5 residues shown by mutagenesis studies to be essential for binding of human CD40 to CD154. hCD154Ckappa was shown to bind pig B cell lines and a proportion of human and pig lymphocytes and further confirmed by staining of COS cells transfected with pig CD40. Conclusions: Recipient human cells bearing CD154 will, therefore, be able to bind donor pig CD40, and these interactions might modulate effector functions and hence influence xenograft survival. Further investigation is necessary to ascertain the exact nature of these interactions and their implications for xenograft survival.

13/7/29 (Item 29 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15473528 BIOSIS NO.: 200000191841
The formation of immunogenic major histocompatibility complex class II-peptide ligands in lysosomal compartments of dendritic cells is regulated by inflammatory stimuli
AUTHOR: Inaba Kayo; Turley Shannon; Iyoda Tomonori; Yamaide Fumiya; Shimoyama Susumu; Reis e Sousa Caetano; Germain Ronald N; Mellman Ira; Steinman Ralph M (Reprint)
AUTHOR ADDRESS: Laboratory of Cell Physiology and Immunology, The Rockefeller University, 1230 York Ave., 405 Bronk Bldg., New York, NY, 10021-6399, USA**USA
JOURNAL: Journal of Experimental Medicine 191 (6): p927-936 March 20, 2000
2000
MEDIUM: print
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: During their final differentiation or maturation, dendritic cells (DCs) redistribute their major histocompatability complex (MHC) class II products from intracellular compartments to the plasma membrane. Using cells arrested in the immature state, we now find that DCs also regulate the initial intracellular formation of immunogenic MHC class II-peptide complexes. Immature DCs internalize the protein antigen, hen egg lysozyme (HEL), into late endosomes and lysosomes rich in MHC class II molecules. There, despite extensive colocalization of HEL protein and MHC class II products, MHC class II-peptide complexes do not form unless the DCs are exposed to inflammatory mediators such as tumor necrosis factor alpha, ***CD40*** ***ligand*** , or lipoploysaccharide. The control of T cell receptor (TCR) ligand formation was observed using the C4H3 monoclonal antibody to detect MHC class II-HEL peptide complexes by flow cytometry and confocal microscopy, and with HEL-specific 3A9 transgenic T cells to detect downregulation of the TCR upon MHC-peptide encounter. Even the binding of preprocessed HEL peptide to MHC class II is blocked in immature DCs, including the formation of C4H3 epitope in MHC class II compartments, suggesting an arrest to antigen presentation at the peptide-loading step, rather than an enhanced degradation of MHC class II-peptide complexes at the cell surface, as described in previous work. Therefore, the capacity of late endosomes and lysosomes to produce MHC class II-peptide complexes can be strictly controlled during DC differentiation, helping to coordinate antigen acquisition and inflammatory stimuli with formation of TCR ligands. The increased ability of maturing DCs to load MHC class II molecules with antigenic cargo contributes to the >100-fold enhancement of the subsequent primary immune response observed when immature and mature DCs are compared as immune

adjuvants in culture and in mice.

13/7/30 (Item 30 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15321415 BIOSIS NO.: 200000039728
Expression of CD40 and its ligand, CD40L, in intestinal lesions of Crohn's disease
AUTHOR: Battaglia Edda; Biancone Luigi; Resegotti Andrea; Emanuelli Giorgio ; Fronda Gian Ruggero; Camussi Giovanni (Reprint)
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JOURNAL: American Journal of Gastroenterology 94 (11): p3279-3284 Nov., 1999 1999
MEDIUM: print
ISSN: 0002-9270
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: OBJECTIVE: Selected mechanisms of the immune system participate in the development of inflammatory bowel disease. Recently, overexpression of the ligand for CD40 (CD40L), a lymphocyte costimulatory molecule, was shown to induce severe inflammatory bowel disease in transgenic mice. In the present study, we examined the expression of CD40 and CD40L on surgical specimens of ileum from 12 patients with Crohn's disease and 10 patients with diverticulitis. METHODS: Several CD40L+ cells were present in the affected tissue of patients with Crohn's disease, whereas few scattered CD40L+ cells were detected in sections of histologically normal ileum, resected distantly from the affected tissue, in patients with diverticulitis and in normal ileum portions obtained from colorectal cancer undergoing extensive surgery. The phenotype of CD40L+ cells was mainly CD4+. RESULTS: In patients with Crohn's disease, several CD40+ cells were detectable in the same areas of lymphocytes expressing CD40L, whereas in patients with diverticulitis, the number of CD40+ cells was significantly lower. Most of the CD40+ cells costained with CD20, thus showing to be B-lymphocytes, and only a few were CD14+ macrophages. Several von Willebrand-positive vessels were also positive for ***CD40*** . In addition, several infiltrating macrophages were found to express B7-1 and B7-2 molecules, the ligands of CD28 and CTLA-4, which cooperate with the CD40-CD40L pathway in lymphocyte activation. Staining of ileal lesions with anti-CTLA-4 ***antibodies*** resulted in ***detection*** of none or very few positive cells. In contrast, in patients with diverticulitis, an enhanced number of B7-1 and B7-2 and CTLA-4 was observed. CONCLUSION: The local accumulation of CD40L+ together with CD40+ cells within intestinal lesions of Crohn's disease suggests the involvement of this co-stimulatory pathway.

13/7/31 (Item 31 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14642989 BIOSIS NO.: 199800437236
Therapy with antibodies against CD40L (CDI54) and CD44-variant isoforms reduces experimental autoimmune encephalomyelitis induced by a proeteolipid protein peptide
AUTHOR: Laman J D (Reprint); Maassen C B M; Schellekens M M; Visser L; Kap M; De Jong E; Van Puijenbroek M; Van Stipdonk M J B; Van Meurs M;

Schwarzler C; Gunthert U
AUTHOR ADDRESS: Div. Immunological Infect. Dis., TNO Prevention Health, PO Box 2215, 2301 CE Leiden, Netherlands**Netherlands
JOURNAL: Multiple Sclerosis 4 (3): p147-153 June, 1998 1998
MEDIUM: print
ISSN: 1352-4585
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Interactions between mononuclear cells are required for the formation of inflammatory infiltrates in the CNS and the activation of cellular effector functions provoking demyelination in MS. Membrane-expressed costimulatory molecules are crucial to such interactions. We therefore investigated whether two costimulatory molecules, CD40L (CD154, expressed on activated CD4-possible T cells) and selected CD44-variant isoforms (expressed on activated CD4-positive T cells), are targets for immunotherapy in MS. The model of experimental autoimmune encephalomyelitis (EAE) induced in SJL-mice by immunization with a peptide derived from the proteolipid protein (PLP139-151) was optimized to address these questions. A previous observation that anti-CD40L (CD154) monoclonal antibodies can effectively prevent EAE in this model was confirmed, and extended by demonstrating that CD40 is expressed by cells of the monocytic lineage infiltrating the spinal cord. In vivo treatment with antibody against the standard isoform of CD44 (CD44s or CD44H) did not affect disease burden. In contrast, combined treatment with antibodies against the isoforms CD44v6, v7 and v10, which are thought to be involved in inflammatory processes, reduced the disease burden considerably. In addition, ***CD44v10*** -expressing cells were ***detected*** in the spinal cord. These data support the idea that CD40-CD40L interactions form a target for immunotherapy of MS, and indicate that cells expressing CD44v6, v7 and/or v10-containing isoforms have such potential as well.

13/7/32 (Item 32 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14434691 BIOSIS NO.: 199800228938
Chronic lymphocytic leukemia B cells can express CD40 ligand and demonstrate T-cell type costimulatory capacity
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AUTHOR ADDRESS: Room C-640, Cornell Univ. Med. Coll., 1300 York Ave., New York, NY 10021, USA**USA
JOURNAL: Blood 91 (8): p2689-2697 April 15, 1998 1998
MEDIUM: print
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Chronic lymphocytic leukemia (CLL) is characterized by a clonal expansion of CD5+ B cells in the peripheral blood. Associated immune aberrations include abnormal Th-cell function and pathogenic autoantibodies. Under most circumstances, CLL B cells do not proliferate in culture and express a limited repertoire of surface antigens, including CD19, CD20, CD23, CD27, ***CD40*** , and CD70. In this report, we demonstrate that freshly isolated B cells from a subset of CLL cases constitutively express CD40 ligand (CD40L, CD154

), a member of the tumor necrosis factor family which is normally expressed by activated CD4+ T cells and mediates T-cell-dependent B-cell proliferation and ***antibody*** production. The degree of ***CD40L*** expression varied considerably among the CLL cases examined. CD40L was detected in purified CLL B cells by immunofluorescence flow cytometry, by RT-PCR, and by immunoprecipitation. To demonstrate that CD40L in the CLL B cells is functional, we used irradiated CLL cells to stimulate IgG production by target, nonmalignant 8 cells in coculture. The CLL B cells induced IgG production by normal B cells to a similar degree as did purified T cells in a process which was partially inhibited by monoclonal antibody to CD40L. This is one of the first reports of CD40L expression in a B-cell tumor. The data suggest that CD40L in the tumor cells may be a factor in the generation of pathologic antibodies by normal B cells in some patients with CLL.

13/7/33 (Item 33 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14386663 BIOSIS NO.: 199800180910
Regulation of cytoplasmic, surface and soluble forms of CD40 ligand in mouse B cells
AUTHOR: Wykes Michelle; Poudrier Johanne; Lindstedt Ragnar; Gray David (Reprint)
AUTHOR ADDRESS: Dep. Immunol., Imperial Coll. Sch. Med., Hammersmith Hosp., Du Cane Rd., London W12 0NN, UK**UK
JOURNAL: European Journal of Immunology 28 (2): p548-559 Feb., 1998 1998
MEDIUM: print
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: CD40 and CD40 ligand (CD40L) form one of most important receptor-ligand pairs that dock during T-B cell interactions as part of T-dependent antibody responses. It has been reported that among other cell types, B cells can express CD40L. Here we show that a large proportion of mouse B cells express CD40L in their cytoplasm, but not on the surface and that this is readily released as a soluble molecule. Thus, in their resting state up to 50% of mouse B cells express CD40L within their cytoplasm and both the proportion of cells expressing and the amount of CD40L is increased by signaling through immunoglobulin (Ig) or CD38. In contrast, T cell-derived signals such as CD40L (anti-CD40) or Th2-type cytokines cause a decrease in CD40L expression that is related to a release of a soluble form of the molecule from the cell. Supernatants from B cells activated with anti-Ig and anti-CD40 contain CD40L in a variety of forms (18 kDa, 33 kDa and 66 kDa) that are readily detectable by immunoprecipitation with CD40-Fcgamma fusion protein (CD40 -Ig) followed by Western blotting with anti-CD40L antibody (MR1). The 33-kDa species is distinct from the 39-kDa membrane-bound molecule found in activated T cells or in resting B cells and appears to be a novel soluble form of CD40L. Inhibition of T cell-independent in vitro stimulation of B cells with CD40-Ig or anti-CD40L suggests that the B cell-derived soluble CD40L or CD40L expressed on the B cell surface can play a positive role in B cell proliferation.

13/7/34 (Item 34 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13925376 BIOSIS NO.: 199799559436
Insert venom immunotherapy induces interleukin-10 production and a Th2-to-Th1 shift, and changes surface marker expression in venom-allergic subjects
AUTHOR: Bellinghausen Iris; Metz Gudrun; Enk Alexander H; Christmann Steffen; Knop Juergen; Saloga Joachim (Reprint)
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JOURNAL: European Journal of Immunology 27 (5): p1131-1139 1997 1997
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The current study was carried out to elucidate the immunoregulatory changes induced by venom immunotherapy (VIT) in bee or wasp allergic subjects. All subjects included in this study had a history of severe systemic allergic reactions to stings of the respective insect as well as positive skin tests with the respective venom or venom-specific IgE in the sera. Parameters assessed in peripheral blood mononuclear cells (PBMC) before and after initiation of VIT (rush therapy reaching a maintenance dose of 100 mu-g venom injected subcutaneously within 1 week) were expression of CD3, CD4, CD8, CD45RA, CD45RO, interleukin (IL)-2 receptor (R)a, IL-4R, IL-12R, Fc-epsilon-RII, CD40, and CD40 ligand (CD40L), cells producing interferon (IFN)-gamma and IL-10 after stimulation with phorbol 12-myristate 13-acetate + ionomycin in the presence of monensin measured by flow cytometry; secretion of IFN-gamma, IL-4, and IL-10 measured by ELISA (IFN-gamma and IL-10 were additionally measured by PCR), and proliferation after stimulation with the respective venom. Significant decreases were observed after VIT for proliferative response to venom and venom + IL-4, IL-4 secretion, Fc-epsilon-RII, ***CD40*** , and ***CD40L*** expression. Significant increases were observed after VIT for IFN-gamma concerning the amount secreted and the number of producing cells, and IL-10. IL-10 was mainly produced by CD4+ cells that were negative for IFN-gamma, but some double-positive (IL-10 and IFN-gamma) cells were always ***detected*** . Addition of blocking anti-IL-10 antibodies, but not isotype control antibodies, prevented down-regulation of proliferation (but not IL-4 secretion) and further enhanced IFN-gamma secretion after VIT. These data indicate that in insect venom allergic subjects, VIT not only induces a rapid shift in cytokine expression from Th2 to Th1 cytokines, but also leads to induction of the immunosuppressive cytokine IL-10, which may be important for the limitation of potentially harmful allergen-specific Th1 responses. The described changes in cytokine expression may be responsible for subsequent increases in allergen-specific IgG and decreases in IgE production, as well as suppressive activity observed in earlier studies.

13/7/35 (Item 35 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13701828 BIOSIS NO.: 199799335888
Induction of cognate and non-cognate T-cell help for B-cell IgE production in relation to CD40 ligand expression
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JOURNAL: International Archives of Allergy and Immunology 111 (4): p 376-384 1996 1996
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LANGUAGE: English

ABSTRACT: Nonactivated, fixed peripheral blood T cells (PBT) from healthy donors or patients with X-linked-hyper-IgM (HIGM) syndrome, or cloned T cells provided effective help for tonsillar B lymphocytes for induction of IgE or other immunoglobulin (1g) isotypes. Helper activity was mediated by staphylococcal superantigens adsorbed to the T cells prior to fixation and required presence of IL-4 in the cultures. We demonstrated that the T cells neither expressed detectable CD40 ligand at the beginning of the superantigen treatment nor 24 h later. Phorbol ester (PMA) plus Ca-ionophore treatment efficiently induced ***CD40L*** . Such T cells did not, however, provide any help for B-cell activation in some experiments or stimulated only low responses in others. ***Antibodies*** against CD2, CD3 and ICAM-1 adsorbed to fixed T cells prior to coculturing inhibited helper activity. A soluble CTLA4 construct was also inhibitory. Our results suggest a pathway of B-cell activation independent of CD40L expressed on T cells.

13/7/36 (Item 36 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13448108 BIOSIS NO.: 199699082168
Human dendritic cells activate T lymphocytes via a CD40: CD40 ligand-dependent pathway
AUTHOR: McLellan Alexander D; Sorg Rudiger V; Williams Lisa A; Hart Derek N J (Reprint)
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JOURNAL: European Journal of Immunology 26 (6): p1204-1210 1996 1996
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The CD40:CD40 ligand (CD40L) interaction provides T lymphocyte-mediated help for B lymphocyte and monocyte function but has also been shown to serve as a co-stimulus for T lymphocyte activation. In this report, we studied the regulation of CD40 expression and its functional relevance for the human dendritic cell (DC) stimulation of T lymphocytes. Only a small subpopulation of directly isolated blood DC expressed CD40. However, CD40 was rapidly up-regulated by culture, and its expression was further enhanced by interleukin (IL)-1-alpha, IL-1-beta, IL-3, tumor necrosis factor-alpha and granulocyte/macrophage-colony-stimulating factor. Expression of ***CD40L*** on DC was not ***detected*** . The proliferation of T lymphocytes in an allogeneic mixed leukocyte reaction, stimulated by blood DC or epidermal Langerhans cells, was significantly reduced in the presence of the CD40 immunoglobulin (CD40Ig) fusion protein or ***CD40L*** monoclonal ***antibodies*** . Cross-linking of ***CD40*** on directly isolated DC with mouse CD40L trimer (mCD40LT) markedly augmented ***CD80*** and ***CD86*** upregulation. Nevertheless, the same

cross-linking mCD40LT inhibited DC stimulated T lymphocyte proliferation. When CD40Ig was added simultaneously with CTLA-4Ig, only minimal and variable additional inhibition of DC-stimulated allogeneic T lymphocyte proliferation and IL-2 secretion was observed, compared to each fusion protein alone. These results suggest that both CD80/CD86-dependent and -independent components of DC-T lymphocyte CD40: CD40L co-stimulation exist and further emphasize that the majority of blood DC have to differentiate or be activated to express co-stimulatory molecules.

13/7/37 (Item 37 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13181573 BIOSIS NO.: 199698649406
CD40 and B cell antigen receptor dual triggering of resting B lymphocytes turns on a partial germinal center phenotype
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JOURNAL: Journal of Experimental Medicine 183 (1): p77-85 1996 1996
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Phenotypic alterations occur when resting human B lymphocytes become germinal center (GC) cells. These include the induction of surface CD38, CD95 (FAS/APO-1), and carboxypeptidase-M (CPM), a recently described GC marker. However, the factors that govern the in vivo induction of these surface molecules on B cells remain unknown. Here, we purified resting (CD38-) human B lymphocytes from tonsils in an attempt to establish culture conditions resulting in the induction of these three GC markers. We show that interferon (IFN) alpha or IFN-gamma, as well as antibodies against the B cell antigen receptor (BCR), could induce CD38 on resting B lymphocytes, a phenomenon further enhanced by CD40 stimulation. Concomitantly, CD95 was upregulated by CD40 ligation and, to a lesser extent, by IFN-gamma. By contrast, CPM expression could be upregulated only through BCR triggering. This CPM induction was specifically enhanced by CD19 or ***CD40*** ligation. ***CD40*** + BCR stimulation of resting B cells with CD40 ligand-transfected fibroblastic cells in the presence of cross-linked anti-BCR monoclonal ***antibodies*** resulted in the coexpression of CD38, CD95, and CPM. As GC cells, these cells also expressed CD71, ***CD80*** (B7.1), and CD86 (B7.2), but not CD24. However, CD10+ or CD44- B cells could not be detected in these culture conditions, suggesting that yet ocher signals are required for the induction of these GC markers. Consistent with a GC phenotype, CD40 + BCR-stimulated cells exhibited reduced viability when cultured for 20 h in the absence of stimulus. These results first demonstrate that cotriggering of resting B cells through BCR and CD40 induces both phenotypic and functional GC features. They also show that IFN and CD19 triggering of resting B cells specifically modulate the expression of GC markers.

13/7/38 (Item 38 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13181555 BIOSIS NO.: 199698649388
Acquisition of CD40 expression during murine B-cell differentiation
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JOURNAL: Scandinavian Journal of Immunology 43 (1): p47-55 1996 1996
ISSN: 0300-9475
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Expression of CD40 on mouse cells was investigated comparing the binding to cells of a monoclonal antibody against CD40, to that of a soluble fusion protein consisting of the extracellular domains of the mouse ***CD40*** - ***ligand*** . The analysis of a series of established cell lines failed to demonstrate expression of CD40 on pro- or pre-B cells, and indicated that CD40 expression was restricted to cells that had undergone productive heavy- and light-chain gene rearrangements, and expressed surface Ig. in cells from normal mice, CD40 first becomes detectable, although at low levels, on a subset of small pre-B-II cells in bone marrow, the levels of CD40 expression increasing thereafter during B-cell maturation. Thus, immature B cells (IgM+ IgD-lo B220-lo) express intermediate levels of CD40, and mature B cells (IgM+ IgD-hi B220-hi) express high levels of CD40. Anatomical location also seems to correlate with the levels of CD40 expression, as B cells expressing the highest levels of CD40 were found in lymph nodes and Peyer's patches.

13/7/39 (Item 39 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13120661 BIOSIS NO.: 199698588494
Rapid induction of a novel costimulatory activity on B cells by CD40 ligand
AUTHOR: Wu Yan; Xu Jianchao; Shinde Sanjay; Grewal Iqbal; Henderson Tanya; Flavell Richard A (Reprint); Liu Yang
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JOURNAL: Current Biology 5 (11): p1303-1311 1995 1995
ISSN: 0960-9822
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: T cells and B cells communicate by direct cell-cell interaction that is crucial to the functioning of the immune system. It is well established that the interaction between B-cell-expressed CD40 and T-cell-expressed CD40 ligand (CD40L) is critical for T-cell-dependent antibody responses, but the role of this interaction in T-cell responses is less clear. In this study, we have used mice with targeted mutations in the genes encoding CD40L or CD28 to investigate how the CD40-CD40L interaction induces on B cells a costimulatory activity that acts in addition to antigen to trigger T-cell growth. Results: We show chat T cells from CD40L-deficient mice induce a substantially reduced costimulatory activity on B cells compared to wild-type T cells, particularly at early time points. Surprisingly, T cells from CD40L-deficient mice induce similar levels of B7-1 and B7-2 as do wild-type T cells. We further show that the ***CD40L*** -mediated induction of costimulatory activity recedes the induction of B7-1, B7-2 and the heat-stable antigen (HSA). ***CD4*** T cells isolated

from the CD28-deficient mice can receive costimulatory activity from CD40L-induced B cells, demonstrating that the induced molecules can costimulate T cells by a CD28-independent mechanism. We have generated a novel monoclonal antibody that inhibits the CD40L-induced costimulatory activity. Expression of the epitope detected by this monoclonal antibody correlates with the induction of the costimulatory activity, and the molecule recognized by the monoclonal antibody is a single chain of around 85 kDa, distinct from B7-1, B7-2, ICAM-1, ICAM-2, ICAM-3, HSA, CD5, integrin and 4-1BB ligand. Conclusions: Our results demonstrate that CD40L is both necessary and sufficient for rapid, T-cell-mediated induction of costimulatory activity on B cells. This costimulatory activity is distinct from B7-1 and B7-2, and is independent of CD28.

13/7/40 (Item 40 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13051690 BIOSIS NO.: 199598519523
CD40 ligand is constitutively expressed in a subset of T cell lymphomas and on the microenvironmental reactive T cells of follicular lymphomas and Hodgkin's disease
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JOURNAL: American Journal of Pathology 147 (4): p912-922 1995 1995
ISSN: 0002-9440
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Although CD40 has been extensively studied in Band T-cell non-Hodgkin's lymphomas (NHLs)/leukemias, and more recently in Hodgkin's disease (HD), little is known about the expression of its ligand (CD40L) in lymphoproliferative disorders other than T-cell NHLs/leukemias. A series of 121 lymphoma/leukemia samples, including 35 cases of HD, 34 T-cell and 39 B-cell NHLs, 2 cases of adult T-cell leukemia/lymphoma, and 11 cases of T-cell acute lymphoblastic leukemia, were evaluated for CD40L expression by immunostaining of frozen tissue sections and flow cytometry with the anti-CD40L monoclonal antibody M90. CD40L was constitutively expressed by neoplastic cells in 15 of 36 (42%) T-cell NHLs/adult T-cell leukemia/lymphomas, almost invariably those displaying the CD4+/CD8-phenotype, whereas no CD40L-expressing tumor cells could be found in B-cell NHL and HD. Among T-cell acute lymphoblastic leukemias, CD40L was detected only on 2 cases displaying a stem-cell-like phenotype. In follicular B-cell lymphomas a large number of CD40L-expressing CD3+/CD4+ T lymphocytes were found admixed with tumor cells within the neoplastic follicles and in their surrounding areas. In the nonfollicular B-cell lymphomas, CD40L-positive CD3+/CD4+ T lymphocytes were few or absent. In all HD subtypes other than the nodular lymphocytic predominance, CD40L-expressing CD3+/CD4+ T lymphocytes were numerous in the HD-involved areas and were mainly located in close proximity to the Reed-Sternberg cells. Our data indicate that in human lymphomas CD40L is preferentially expressed by a restricted subset of T-cell lymphomas, mostly with ***CD4*** immunophenotype. Finally, we have provided morphological evidence that CD40L may play an important role in the cell contact-dependent interaction of tumor B-cells (CD40+) within the neoplastic follicles or Reed-Sternberg cells (CD40+) in HD-involved areas and the microenvironmental CD3+/

CD4 +/ ***CD40L*** + T lymphocytes.

13/7/41 (Item 41 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12799797 BIOSIS NO.: 199598267630
A subset of CD4+ memory T cells contains preformed CD40 ligand that is rapidly but transiently expressed on their surface after activation through the T cell receptor complex
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JOURNAL: Journal of Experimental Medicine 181 (4): p1293-1301 1995 1995
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Signaling through surface CD40 is essential for selecting B cells that have mutated their immunoglobulin variable region genes in germinal centers and is an important signal in the early stages of antibody responses to T cell-dependent antigens. It is shown that a subset of CD45RO+, CD4+ T cells isolated from human tonsil contains preformed 30-35-kD ligand for CD40. This is expressed on their surfaces within 5 min of their antigen-receptor complexes interacting with CD3-epsilon ***antibodies*** bound to ox erythrocytes. This surface expression does not require de novo protein synthesis and lasts for only 1-2 h. Preformed CD40 ligand (CD40L) was not detected in any CD4+ CD45RA+ T cells, but gt 90% of all CD4+ T cells from the tonsil can be induced to express large amounts of CD40L on culture with phorbol myristate acetate and the calcium ionophore ionomycin. This expression of ***CD40L*** starts between 1 and 2 h, peaks at 6 h, and remains at a high level for gt 20 h. It is totally prevented by adding a concentration of cycloheximide that inhibits CD25 synthesis by these activated cells. While CD3-epsilon antibody bound to ox red cells is a good inducer of surface expression of CD40L, it is a much less potent inducer of CD40L synthesis than phorbol myristate acetate with ionomycin. Immunohistological analysis of tonsil sections shows that cells containing CD40L are located mainly in the outer zone of germinal centers and the margins of the T zones that are rich in dendritic cells (interdigitating cells). The distribution of these cells is consistent with: (a) their interaction in T zones with B cells that have taken up and processed antigen and (b) their involvement in B cell selection in germinal centers.

13/7/42 (Item 42 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12719124 BIOSIS NO.: 199598186957
Gamma/delta T Lymphocytes Express CD40 Ligand and Induce Isotype Switching in B Lymphocytes
AUTHOR: Horner Anthony A; Jabara Haifa; Ramesh Narayanaswamy; Geha Raif S (Reprint)
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JOURNAL: Journal of Experimental Medicine 181 (3): p1239-1244 1995 1995

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RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: T cells expressing gamma/delta T cell receptors home to epithelial tissue and may play a role in immunity to infectious agents and foreign antigens. In an effort to understand the role of gamma/delta T cells in directing B cell responses, we investigated the capacity of human gamma/delta T cells to express CD40 ligand (CD40L) and to drive immunoglobulin (Ig) isotype switching in B cells. A multiple step purification procedure resulted in the recovery of highly pure populations of peripheral blood CD4-CD8- gamma/delta T cells. Neither ***CD40L*** surface expression nor ***CD40L*** mRNA were detected in unstimulated gamma/delta T cells. Stimulation with phorbol ester and ionomycin induced CD40L mRNA and surface CD40L expression by gamma/delta T cells. Both the percentage of ***CD40L*** + cells and the cell surface density of CD40L were significantly lower in gamma/delta T cells compared to unselected T cells. We further demonstrated that in the presence of neutralizing monoclonal antibody to interferon gamma (IFN-gamma), gamma/delta T cells could induce IgE synthesis in B cells, albeit to a lesser extent than unselected T cells. Furthermore, IgE synthesis driven by gamma/delta T cells was inhibited by monoclonal antibody to CD40L. These observations demonstrate that activated gamma/delta T cells express CD40L and can induce isotype switching in B cells.

13/7/43 (Item 43 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12671998 BIOSIS NO.: 199598139831
A cytotoxic CD40/p55 tumor necrosis factor receptor hybrid detects CD40 ligand on herpesvirus saimiri-transformed T cells
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JOURNAL: European Journal of Immunology 25 (1): p80-86 1995 1995
ISSN: 0014-2980
DOCUMENT TYPE: Article
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LANGUAGE: English

ABSTRACT: The B cell activation molecule CD40 and the p55 tumor necrosis factor receptor (p55TNFR) belong to the same family of structurally conserved proteins. We constructed a chimeric receptor consisting of the CD40 extracellular and transmembrane domains and the p55TNFR intracellular domain. This receptor hybrid retained the biological activity and the ligand specificity of the respective wild-type receptor domains. Thus it exerted a marked cytotoxic effect in three different transfected cell lines after activation not only with anti-CD40 antibody but also with CD40 ligand (CD40L) in soluble and membrane-bound forms. Using hybrid-transfected baby hamster kidney cells we demonstrated that herpesvirus saimiri-transformed human CD4+ T lymphocytes constitutively express bioactive CD40 ***ligand*** on their surface. The hybrid receptor-based assay was highly specific for CD40 activating reagents and more sensitive than an assay measuring ***CD40*** -mediated B cell rescue from apoptosis. Hence CD40/p55TNFR transfectants may be useful for dissecting CD40L

-mediated events in T-B cell interactions, and also to detect a defective ***CD40L*** molecule in putative hyper-IgM syndrome patients.

13/7/44 (Item 44 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12428391 BIOSIS NO.: 199497449676
Decreased expression of the ligand for CD40 in newborn lymphocytes
AUTHOR: Fuleihan Ramsay (Reprint); Ahern Deborah; Geha Raif S
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JOURNAL: European Journal of Immunology 24 (8): p1925-1928 1994 1994
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RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Immune responses in newborn lymphocytes show a defect in isotype switching from IgM to IgG and IgA. Immunoglobulin isotype switching in B lymphocytes requires a contact-dependent signal from T lymphocytes which is delivered by the ligand for the B cell surface antigen CD40. We investigated the capacity of newborn lymphocytes to express the CD40 ligand and to undergo CD40 ligand-dependent immunoglobulin isotype switching. After stimulation by phorbol ester and ionomycin, newborn lymphocytes expressed markedly decreased amounts of CD40 ligand on their surface compared to normal adult lymphocytes. Northern blot analysis of mRNA derived from activated cord blood lymphocytes also revealed markedly decreased amounts of CD40 ligand mRNA. Decreased expression of CD40 ligand in newborn lymphocytes was associated with a severely decreased ability to undergo T cell-dependent immunoglobulin isotype switching. Newborn lymphocytes synthesized little or no detectable IgE in response to T cell-dependent stimulation by interleukin-4 but synthesized IgE in response to T cell-independent stimulation by CD40 monoclonal ***antibody*** and interleukin-4. These results indicate that decreased expression of CD40 ligand in newborn lymphocytes may be the underlying cause of deficient immunoglobulin isotype switching in newborns.

13/7/45 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

0082512259 EMBASE No: 2008320396
CD4 SUP +T cells in CIKs (CD4 SUP + CIKs) reversed resistance to fas-mediated apoptosis through CD40/CD40L ligation rather than IFN-gamma stimulation
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LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 28

Background: Cytokine-induced killer cells (CIKs) are nonspecific antitumor effectors with superior advantages. CD4 SUP + CIKs can induce Fas-dependent apoptosis in sensitive Raji cells. Here, a Fas-dependent apoptosis was detected in resistant breast cancer MDA-MB-231 cells, and underlying mechanisms were discriminated. Methods: Amplification of CIKs and purification of CD4 SUP + CIKs were performed in 15 patients with malignant solid tumors. The expression of ***CD40L*** and soluble cytokines in ***CD4*** SUP + CIKs were analyzed. The apoptotic rates of tumor cells and the expression of Fas on membranes were detected using flow cytometry assay. The specific blocking ***antibodies*** against FasL, CD40L, and interferon-gamma (IFN-gamma) were added to abolish their effects. The changes of 4 apoptosis-related genes (Bcl-2, Bax, Fas-associating protein with death domain [FADD], and FLICE inhibitory protein [c-FLIP]) in MDA-MB-231 cells cocultured with CD4 SUP + CIKs were measured by real-time quantitative reverse-transcriptase polymerase chain reaction after 6 hours and 24 hours with or without blocking antibodies. Results: Upregulated expression of membrane-attached CD40L and dramatically increased secretion of soluble CD40L and IFN-gamma were identified in CD4 SUP + CIK. The susceptibility to Fas-mediated apoptosis of insensitive MDA-MB-231 cells was elevated after being pretreated with supernatants from CD4 SUP + CIK. After coculture with CD4 SUP + CIK, apoptosis in MDA-MB-231 cells paralleled with enhanced expression of Fas was blocked fully by either anti-FasL or anti-CD40L, but only partly by anti-IFN-gamma antibodies. The anti-CD40L monoclonal antibody (McAb) rather than anti-IFN-gamma McAb induced significant increase of c-FLIP, which negatively correlated with the apoptosis observed in MDA-MB-231 cells. Conclusions: Apoptosis in MDA-MB-231 cells induced by CD4 SUP + CIK is Fas-dependent. The reversion of Fas resistance is mediated through CD40/CD40L ligation rather than IFN-gamma stimulation by inhibiting synthesis of c-FLIP.

13/7/46 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

0082178743 EMBASE No: 2007592369
Germinal center B cells are dispensable in prion transport and neuroinvasion

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NUMBER OF REFERENCES: 54

Transmissible spongiform encephalopathies (TSEs) are fatal

neurodegenerative diseases of animals and humans. Many TSEs are initiated by prion replication in the lymphoreticular system (LRS). The cellular and molecular prerequisites for prion trafficking within the LRS are not fully understood. Here we have manipulated CD40 and its ligand to investigate whether genetic or pharmacological ablation of germinal center B cells (GCBs), which migrate into and out of germinal centers, influences prion pathogenesis. In contrast to previous reports, no alteration of prion pathogenesis was detected in mice lacking CD40L and in mice treated with anti- ***CD40L*** ***antibodies*** . These results suggest that GCBs alone do not impact peripheral splenic prion transport, replication efficiency, or neuroinvasion, and point to other mechanisms affecting prion transport from lymphoreticular sites of replication to the nervous system.

13/7/47 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

0081063500 EMBASE No: 2006123577

The CD40/CD40 ligand system is expressed in the cutaneous lesions of erythema multiforme and Stevens-Johnson syndrome/toxic epidermal necrolysis spectrum

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NUMBER OF REFERENCES: 36

Background: Erythema multiforme (EM) and Stevens-Johnson syndrome (SJS)/toxic epidermal necrolysis (TEN) are determined by a dysregulation of cellular immunity. Objectives: To evaluate the effector role of cellular immunity and the involvement of the CD40/CD40 ligand (CD40L) system in the pathogenesis of EM and SJS/TEN. Methods: Biopsy specimens from eight patients with EM and six with SJS/TEN were stained for immunohistochemical examination using the alkaline phosphatase/antialkaline phosphatase method. The monoclonal antibodies used included those to CD1a, CD4, CD8, CD40, CD40L, CD68, Fas, Fas ligand (FasL) and myeloperoxidase. Results: The cellular infiltrate in both EM and SJS/TEN lesions was composed mainly of T lymphocytes and CD68+ macrophages. We also ***detected*** large amounts of neutrophils. Fas and FasL were very highly expressed in SJS and TEN, but weakly in EM. ***CD40*** staining was strong in all tissue sections; there were numerous CD40L+ cells in SJS/TEN but much fewer in EM. Conclusions: Activated T lymphocytes and macrophages, but also neutrophils, are presumably the main triggers of mucocutaneous damage in the SJS/TEN disease spectrum. The Fas/FasL system is significantly expressed in SJS/TEN lesions, but not in EM, where this apoptotic pathway presumably does not play a pivotal role in the epidermal damage. We suggest that the CD40/CD40L system may represent an important

pathway of induction of SJS/TEN lesions, while in EM it would contribute to the immunoinflammation only as a second-line mechanism. (c) 2005 British Association of Dermatologists.

13/7/48 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

0080393439 EMBASE No: 2005037586
The role of CD40-CD154 interactions in autoimmunity and the benefit of disrupting this pathway
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Autoimmunity (Autoimmunity) (United Kingdom) September 1, 2004, 37/6-7 (457-464)
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NUMBER OF REFERENCES: 53

Many tissue injuries and immune mediated pathologies such as graft allo-rejections were found to involve CD40-CD40 ligand (***CD40L*** , ***CD154***) signaling pathway. The disruption of this pathway in many animal models led to the improvement of graft survival in these models. ***CD40*** - ***CD154*** interactions were also shown to play a significant role in the maintenance of autoimmunity, and the production of auto- ***antibodies*** in systemic lupus erythematosus (SLE). High-level expression of CD154 has been detected on T cells from patients with active SLE, rheumatoid arthritis (RA) and other autoimmune diseases, indicating that such cells could account for the high-level expression of immune accessory molecules on B cells of patients with active disease. An increased serum level of soluble CD154 was also reported in SLE, RA, and Sjogren's disease in correlation with the relevant auto-antibodies and with the clinical disease activity. Anti-CD154 antibody therapy prevents auto-antibody production and renal immune complex deposition in lupus nephritis, indicating that disruption of this pathway could be a beneficial treatment in SLE. However, the etiology of the higher than expected number of thromboembolic events in anti-CD154 treated SLE patients should be investigated and preventive measures should be considered. (c) 2004 Taylor & Francis Ltd.

13/7/49 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

0080014361 EMBASE No: 2004199525
Coexpression of CD40 and CD40L on B lymphoma and carcinoma cells: An autocrine anti-apoptotic role
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DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 34

To evaluate a possible autocrine role of CD40L, the expression and functional activity of CD40L on NHL and breast carcinoma cell lines were investigated. Using flow cytometry, CD40 was consistently detectable at the surface of all 5 NHL cell lines tested. CD40L expression was detectable at the surface of DAUDI (54%, MFI 47) and BJAB (12%, MFI 32) cell lines, and marginally on the RAJI cell line (7%, MFI 30), while 4 of 5 NHL cell lines (DAUDI, RAJI, BJAB, BL70) had ***detectable*** CD40L mRNA. CD40 was expressed on T47D and BT20 breast carcinoma cell lines while CD40L was detectable on T47D (93%, MFI 137) only. Both BT20 and T47D had detectable CD40 mRNA, while CD40L mRNA was detectable only in the T47D cell line. CD40, but not CD40L, was detectahle on 6 renal, 1 prostatic and 1 colon carcinoma cell lines. CD40L expressed on tumor cells was functional, as shown by its capacity to decrease drug-induced apoptosis on CD40 expressing NHL and breast carcinoma cell lines, while irradiated CD40L negative cell line (BT20) had no effect. Blocking ***CD40L*** antibody abrogated the protective effect of irradiated CD40L positive T47D cell line against drug-induced apoptosis on BL70 cell line, confirming that CD40L is functional in the DAUDI and T47D cell lines. Importantly, blocking CD40L antibody increased drug-induced apoptosis in CD40L positive cell lines but had no effect on the CD40L negative cell lines. ***CD40L*** is expressed on ***CD40*** positive B NHL and breast carcinoma cell lines and induces an autocrine antiapoptotic signal when cells are exposed to cytotoxic agents. (c) 2004 Taylor & Francis Ltd.

13/7/50 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE

0079624502 EMBASE No: 2003332562
Human autologous dendritic cell-glioma fusions: Feasibility and capacity to stimulate T cells with proliferative and cytolytic activity
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LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 31

Gliomas are the most common primary neoplasm of the central nervous

system. The failure of conventional treatment modalities to improve outcome over the last two decades has led to interest in alternative treatment modalities. Dendritic cell (DC)-based immunotherapy has utilized DC pulsed with tumor lysate or peptide to induce an anti-tumor immune response mediated largely by CD8 T cells. While this has been effective in preclinical studies, clinical efficacy remains unproven. Recently, hybrid cells produced by fusions of tumor and autologous DC have demonstrated remarkable efficacy for stimulating an anti-tumor immune response in both preclinical and clinical studies of extra-cranial neoplasms. The advantage of generating such hybrid cells is that the entire cellular material of the tumor is processed and presented in both endogenous and exogenous pathways. This leads to activation of both MHC class I restricted CD8 cells as well as MHC class II restricted CD4 T cells. Here, we examined in vitro T cell stimulatory capacity of autologous human DC-glioma fusion in comparison to DC loaded with apoptotic glioma. DC fused with autologous tumor or loaded with apoptotic tumor cells (DC/apo) were first used to stimulate autologous non-adherent peripheral blood mononuclear cells (PBMC), in vitro. The PBMC were then examined for phenotype (CD3, CD4, CD8) and intracellular IFN-gamma using flow cytometry. Lymphocyte proliferation and cytolytic responses were also assessed. Lymphocytes stimulated in vitro with fusion or DC/apo cells showed significantly enhanced cytotoxicity and proliferation against autologous tumor cells compared with PBMC stimulated with tumor cells or DC alone. Both strategies had similar efficacy. Tumor-cytolytic responses were enhanced by the addition of CD40 ligand (CD40L), and partially blocked by anti-MHC class I
antibody . Flow cytometric analysis ***detected*** CD3 SUP + ***CD8***
SUP + T cells, which also stained positive for intracellular IFN-gamma. The study suggests that DC/glioma fusion and DC/apo have comparable efficacy for stimulation of CTL with cytolytic and proliferative activity against human malignant gliomas. These findings may have implications for future studies of DC-based immunotherapy in malignant gliomas.

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DIALOG(R)File 73:EMBASE

0079258619 EMBASE No: 2002422927
CD40 activation as potential tool in malignant neoplasms
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LANGUAGE: English SUMMARY LANGUAGE: English
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Background: CD40, a cell surface molecule, is expressed on B-cell malignancies and many different solid tumors. It is capable of mediating diverse biological phenomena such as the induction of apoptosis in tumors and stimulation of the immune response. It has thus been studied as a possible target for antitumor therapy. The general aim of this review is to focus the attention of clinical oncologists on the involvement of CD40 in tumors and the rationale of CD40-activation-based therapies in new,

biologically oriented antitumor protocols. Methods: A Medline review of published papers about the role of CD40 activation in cancer therapy. Results: Many authors have shown that CD40 activation promotes apoptotic death of tumor cells and that the presence of the molecule on the surface of carcinoma lines is an important factor in the generation of tumor-specific T-cell responses that contribute to tumor cell elimination. The CD40 ligand (CD40L) is the natural ligand for CD40; it is expressed primarily on the surface of activated T lymphocytes. Preclinical studies suggest that ***CD40*** - ***CD40L*** interaction could be useful for cytotoxicity against CD40-expressing tumors and for immune stimulation. Tumor inhibition was observed when tumor cells were treated with agonistic anti-CD40 monoclonal antibodies or with the soluble form of ***CD40L*** . The results of the first phase I clinical trial to treat cancer patients with subcutaneous injection of recombinant human ***CD40L*** have been recently reported. Immunohistochemical studies have revealed that detection of CD40 in primary cutaneous malignant melanoma and lung cancer may have a negative prognostic value. Interestingly, up-regulation of ***CD40*** was observed in the tumor vessels of renal carcinomas and Kaposi's sarcoma, suggesting possible involvement of CD40 in tumor angiogenesis. Recently, it has also been shown that CD40 engagement, on endothelial cells induces in vitro tubule formation and expression of matrix metalloproteinases, two processes involved in the neovascularization and progression of tumors. Conclusions: CD40 activation represents an exciting target for hematological malignancies and solid tumors expressing the molecule, but its functional role in cancer development still remains unclear and controversial.

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DIALOG(R)File 73:EMBASE

0078562743 EMBASE No: 2001168884
CD40 ligand expression in Mycobacterium bovis BCG infection and its regulation by cytokines: A direct role of interleukin 12
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NUMBER OF REFERENCES: 19

Background: Activation and clonal expansion of T cells require not only the recognition of processed antigen on the surface of the antigen-presenting cell (APC) by T-cell receptor (TCR), but also involve co-stimulatory signals that are provided by the simultaneous engagement of cell surface molecules expressed by both the APC and the T cell. Interaction between CD40 and its ligand (CD40L) is known to mediate host immune response and T-cell-mediated effector functions in mycobacterial infections in mice. In this work, we investigated the capacity of Mycobacterium bovis (M. bovis) BCG to induce the expression of CD40L on

human T cells. Methods: Human cells were obtained from healthy adults by centrifugation using Ficoll/Hypaque. Cells (1 x 10 SUP 6) were incubated in RPMI medium with BCG. After incubation at 37(deg)C in 5% CO SUB 2 atmosphere for 40 h, cells were collected and double-stained with anti- ***CD40L*** -PE and anti- ***CD4*** -FITC or anti- ***CD8*** -FITC mAb. The quantification of positively stained population was based on samples stained with isotype control ***antibodies*** analyzed on a FACScan. Results: M. bovis BCG stimulation induced a significant amount of CD40L expression on CD4+ T cells, while CD40L was not significantly ***detected*** on human ***CD8*** + T cells. The highest CD40L expression on BCG-activated T cells in synergism with interleukin-12 endogenous occurred after a 40-h cell culture with a dose of 10 mug/mL of BCG (84.86 +/- 11.77; mean +/- standard deviation [SD]). This CD40L expression on BCG-activated human T cells was significantly inhibited by anti-IL-12 mAb (5.08 +/- 1.7; mean +/- SD). In contrast, anti-IFN-gamma and anti-IL-2 mAb did not have an important role in this expression. Conclusions: These results indicate that the capacity of BCG to induce CD40L expression on human cells represents a novel mechanism underlying the regulation of cellular responses against tuberculosis. Furthermore, the results showed a direct role of IL-12 to enhance the expression of CD40L on BCG-activated human cells. Copyright (c) 2001 IMSS.

13/7/53 (Item 9 from file: 73)
DIALOG(R)File 73:EMBASE

0078517595 EMBASE No: 2001123685
Analysis of the CD40/CD40L role in the sustenance of alloreactive antibody production
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LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 54

CD40 ligand (CD40L) is important for T/B lymphocyte interaction. To understand the cellular basis of humoral allosensitization we, therefore: (1) measured CD40L protein and gene expression in sensitized and non-sensitized uremic unactivated peripheral CD4 SUP + T lymphocytes; (2) studied the impact of blocking the CD40/CD40L pathway on alloreactive antibody (allo-Ab) production by engrafted sensitized PBLs into severe combined immunodeficient (SCID) mice after in vitro preactivation with IL SUB 2/LPs/HLA class II allopeptides and adjuvants as a potent stimulus to produce allo-Ab (Shoker et al. Transplantation 1999;68;1188); and (3) studied the modifying effect of CD40/CD40L blockade on T helper type I and

II cytokine gene expression in the respective mice spleen. The CD40L protein was measured by flow cytometry and the gene expression was measured by quantitative RT-PCR. Alloreactive ***antibodies*** (alo-Abs) produced by sensitized PBLs engrafted into SCID mice with and without blockade of the CD40 receptor were measured by the PRA-STAT ELISA method. The modifying effects of CD40 blocking on allo-Ab production and cytokine gene expression by the engrafted cells measured by RT-PCR were then compared. There was no detectable CD40L protein expression in either the uremic or the control groups. The ***CD40L*** gene expression of 0.04 +/- 0.02 attomoles (aM) in the sensitized group was significantly higher than in the non-sensitized patients (0.009 +/- 0.007 aM, P < 0.0001) or the control CD4 SUP + T cells (0.016 +/- 0.004 aM, P < 0.001). Blockade of the CD40 receptor abrogated the production of allo-Ab antibodies by the engrafted sensitized cells in 60% of the tested mice (n = 10); decreased the mean +/- S.D. optic density of allo-Ab to 0.1 +/- 0.13 and the mean +/- S.D. PRA to 12 +/- 16). In the presence of the control Ab, allo-Ab production in SCID sera was present in 100% of the 10 SCID mice tested; the mean +/- S.D. PRA was 75 +/- 20, and the mean +/- S.D. OD activity was 0.412 +/- 0.17. All cytokine genes were, otherwise, expressed in the presence or absence of CD40 blockade. The results suggest a potential role of an enhanced CD40/CD40L interaction in the sustenance of alloreactive antibody production without significant deviation to T helper-like I or II responses. Blocking the CD40/CD40L pathway may have a potential therapeutic benefit to treat sensitized uremic patients.

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0078283506 EMBASE No: 2000333096
Expression of CD40 and CD40 ligand in Bowen's disease and squamous cell carcinoma
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European Journal of Dermatology (Eur. J. Dermatol.) (France) October 4, 2000, 10/6 (439-442)
CODEN: EJDEE ISSN: 1167-1122
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 19

CD40 is a member of the tumor necrosis factor receptor super-family expressed by B cells, monocytes, dendritic cells, epithelial cells and hematopoietic progenitor cells. ***CD40*** has recently been reported to be expressed on several epidermal tumors as well. ***CD40*** on epidermal tumor cells interacts with lymphocytes expressing ligand for CD40 (CD40L) or monoclonal antibodies against CD40 with a significant decrease in proliferation. In this study, we examined the expression of CD40 and CD40L in Bowen's disease and squamous cell carcinoma (SCC). ***CD40*** immunoreactivity was observed in a significantly lower proportion of tumor cells from SCC than from Bowen's disease. ***CD40L*** mRNA expression was detected in Bowen's disease and SCC by reverse transcriptase polymerase chain reaction (RT-PCR). ***CD40*** - ***CD40L*** interactions in epidermal tumors may play a role in the proliferation, and

the lack of CD40 in tumor cells from SCC might be involved in the mechanisms of escape from the growth inhibitory effect.

13/7/55 (Item 11 from file: 73)
DIALOG(R)File 73:EMBASE

0077542733 EMBASE No: 1999028864
CD40L (CD 154) expression in human liver allografts during chronic ductopenic rejection
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Liver Transplantation and Surgery (Liver Transplant. Surg.) (United States) February 22, 1999, 5/1 (1-7)
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DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 42

The CD40-CD40L (CD154) interaction plays a pivotal role in the effector mechanisms of allograft rejection. Blockade of the CD40/CD40L costimulatory pathway prevents the development of chronic allograft rejection in several animal transplant models. The relevance of in situ CD40 and CD40L expression in human liver allografts was assessed by immunohistochemistry during ductopenic chronic rejection (CR). In CR allograft specimens (n = 8), marked ***CD40L*** expression was detected on Kupffer cells (KCs) and sinusoidal macrophages with a unique centrilobular distribution (P < .001). The CD40L+ KCs and macrophages were shown to be CD68+ after immunohistochemical analysis of serial sections with anti-CD68 monoclonal ***antibody*** . Moderate staining of vascular and sinusoidal endothelial cells and mononuclear infiltrates was observed in some CR cases. These findings were in contrast to the absence of CD40L expression in controls (n = 11) consisting of stable liver allograft and normal liver tissue specimens. Only occasional CD40 expression in some cases of CR and controls was observed. In CR, CD40L (CD154) expression is manifested on KCs and macrophages. The present novel data show another important cellular source of CD40L expression and suggest a potential role of KCs/macrophages and CD40/CD40L costimulatory interactions in the pathogenesis of chronic rejection ductopenic liver allograft.

13/7/56 (Item 12 from file: 73)
DIALOG(R)File 73:EMBASE

0077391132 EMBASE No: 1998301549
Therapy with antibodies against CD40L (CD154) and CD44-variant isoforms reduces experimental autoimmune encephalomyelitis induced by a proteolipid protein peptide
Laman J.D.; Maassen C.B.M.; Schellekens M.M.; Visser L.; Kap M.; De Jong E.; Van Puijenbroek M.; Van Stipdonk M.J.B.; Van Meurs M.; Schwarzler C.; Gunthert U.

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NUMBER OF REFERENCES: 25

Interactions between mononuclear cells are required for the formation of inflammatory infiltrates in the CNS and the activation of cellular effector functions provoking demyelination in MS. Membrane-expressed costimulatory molecules are crucial to such interactions. We therefore investigated whether two costimulatory molecules, CD40L (CD154, expressed on activated CD4-possible T cells) and selected CD44-variant isoforms (expressed on activated CD4-positive T cells), are targets for immunotherapy in MS. The model of experimental autoimmune encephalomyelitis (EAE) induced in SJL-mice by immunization with a peptide derived from the proteolipid protein (PLP139-151) was optimized to address these questions. A previous observation that anti-CD40L (CD154) monoclonal antibodies can effectively prevent EAE in this model was confirmed and extended by demonstrating that CD40 is expressed by cells of the monocytic lineage infiltrating the spinal cord. In vivo treatment with antibody against the standard isoform of CD44 (CD44s or CD44H) did not affect disease burden. In contrast, combined treatment with antibodies against the isoforms CD44v6, v7 and v10, which are thought to be involved in inflammatory processes, reduced the disease burden considerably. In addition, ***CD44v10*** -expressing cells were ***detected*** in the spinal cord. These data support the idea that CD40-CD40L interactions form a target for immunotherapy of MS, and indicate that cells expressing CD44v6, v7 and/or v10-containing isoforms have such potential as well.

13/7/57 (Item 13 from file: 73)
DIALOG(R)File 73:EMBASE

0076846513 EMBASE No: 1997139570
Insect venom immunotherapy induces interleukin-10 production and a Th2-to-Th1 shift, and changes surface marker expression in venom-allergic subjects

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CODEN: EJIMA ISSN: 0014-2980
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 52

The current study was carried out to elucidate the immunoregulatory changes induced by venom immunotherapy (VIT) in bee or wasp allergic subjects. All subjects included in this study had a history of severe

systemic allergic reactions to stings of the respective insect as well as positive skin tests with the respective venom or venom-specific IgE in the sera. Parameters assessed in peripheral blood mononuclear cells (PBMC) before and after initiation of VIT (rush therapy reaching a maintenance dose of 100 mug venom injected subcutaneously within 1 week) were expression of CD3, CD4, CD8, CD45RA, CD45R0, interleukin (IL)-2 receptor (R)alpha, IL-4R, IL-12R, FcepsilonRII, CD40, and CD40 ligand (CD40L), cells producing interferon (IFN)-gamma and IL-10 after stimulation with phorbol 12-myristate 13-acetate + ionomycin in the presence of monensin measured by flow cytometry; secretion of IFN-gamma, IL-4, and IL-10 measured by ELISA (IFN-gamma and IL-10 were additionally measured by PCR), and proliferation after stimulation with the respective venom. Significant decreases were observed after VIT for proliferative response to venom and venom + IL-4, IL-4 secretion, FcepsilonRII, ***CD40*** , and ***CD40L*** expression. Significant increases were observed after VIT for IFN-gamma concerning the amount secreted and the number of producing cells, and IL-10. IL-10 was mainly produced by CD4 SUP + cells that were negative for IFN-gamma, but some double-positive (IL-10 and IFN-gamma) cells were always ***detected*** . Addition of blocking anti-IL-10 ***antibodies*** , but not isotype control antibodies, prevented down-regulation of proliferation (but not IL-4 secretion) and further enhanced IFN-gamma secretion after VIT. These data indicate that in insect venom allergic subjects, VIT not only induces a rapid shift in cytokine expression from Th2 to Th1 cytokines, but also leads to induction of the immunosuppressive cytokine IL-10, which may be important for the limitation of potentially harmful allergen-specific Th1 responses. The described changes in cytokine expression may be responsible for subsequent increases in allergen-specific IgG and decreases in IgE production, as well as suppressive activity observed in earlier studies.

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DIALOG(R)File 73:EMBASE

0076434870 EMBASE No: 1996111086
CD40 expression by human peripheral blood eosinophils
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April 1, 1996, 97/7 (1761-1766)
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In this study, we have investigated CD40 expression in human peripheral blood eosinophils and in human chronically inflamed nasal tissues, i.e., nasal polyps. We show by both reverse transcriptase-PCR and Northern blot analysis that eosinophils from allergic subjects express human CD40 mRNA. We also show that constitutive CD40 mRNA expression in eosinophils could be upregulated by exposure to IgA immune complexes and downregulated by IL-10 and the synthetic steroid budesonide. In addition, we demonstrate that eosinophils express CD40 protein by flow cytometry. Such expression is biologically functional as cross-linking CD40 with CD40 mAbs enhances eosinophil survival in a dose-dependent fashion; in addition, CD40 ligation

stimulates eosinophils to release GM-CSF. CD40-mediated eosinophil survival was largely inhibited by an anti-GM-CSF neutralizing antibody suggesting GM- CSF involvement in the survival enhancing mechanism. CD40 mRNA was also detected in total RNA extracted from nasal polyp tissues but not in RNA isolated from normal nasal mucosa (inferior turbinate); by immunohistochemistry, we were able to detect immunoreactive CD40 protein in a variety of cell types in the polyp stroma, but primarily in eosinophils. These observations suggest previously unforeseen interactions between eosinophils and cells expressing the CD40 ligand and, thus, novel pathways by which eosinophils may contribute to the regulation of airway inflammation.
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